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THE
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THE PLACE OF THE OBLITERATIVE OPERATION IN FRONTAL SINUS SURGERY.*†‡

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I. INTRODUCTION.

This paper is presented as a study of the problem of frontal sinus surgery. The literature is reviewed. Three illustrative cases are reported from a series of 17 cases of frontal sinus infection in which surgery was performed. In 11 cases an obliterative operation of the frontal sinuses was done. In two cases a gold Ingals' tube was permanently inserted. In four cases a modified Lynch type operation was done. One case from each of these three groups is reported in detail. An analysis of these cases is presented in an attempt to arrive at a rational basis for the proper type of surgical treatment of frontal sinus disease.

II. HISTORICAL REVIEW.

The literature on frontal sinus surgery is voluminous and controversial. Time and again the pendulum swings toward

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and away from radical surgery. Ogston,[1] in 1884, was the first to report opening the frontal sinuses for the drainage of infection. Riedel,[2] in 1898, first described the complete obliterative operation on the frontal sinus. This apparently attracted little attention in this country. In 1903, Killian[3] described his operation for frontal sinus disease. In the classical Killian operation, the entire floor and all of the front wall of the frontal sinus, except a bony bridge to preserve the orbital ridge, are removed. The Killian operation attracted widespread attention and became very popular in this country.

In 1905, there were Symposiums on frontal sinus disease at the Annual Meetings of both the Americal Laryngological, Rhinological and Otological Society, Inc., and the American Laryngological Society. There were several other articles published that same year. There was great interest in surgery of the frontal sinus. Coffin[4] discussed various external operations which were being developed and classified them in three types: *1.* the Ogston-Luc with a relatively small opening into the sinus through the anterior wall, *2.* the Killian operation, *3.* the open method, attempting to obliterate the sinus by packing through an open wound (Kuhnt-Coakley). Although he performed radical operations on the frontal sinus, he decried "frenzied surgery." He also mentioned the desirability of X-rays which were just coming into use. Richards[5] reported 14 cases, most of which had a stormy course with several operations, usually starting with a Killian operation and ending up with external obliteration. Coakley[6] discussed frontal sinusitis, acute and chronic, and used a radical obliterating type of operation with packing of the open wound. In the acute cases he first infracted the middle turbinate or removed its anterior end. If improvement did not follow, he performed a radical operation promptly. The severe cases were desperate and frequently died. The idea of waiting until the acute symptoms had subsided before radical surgery was done had not been presented. In his chronic cases the mortality rate was much lower. At the same time other authors strongly urged reliance on intranasal treatment. Casselberry[7] was very emphatic, citing the high mortality and the great

deformity of the radical operation. Ingals[8] first described his flanged, expanding gold tube, which is still in use today. As he originally presented it, it was for intranasal introduction with a special introducer and was left in place only temporarily.

The first wave of enthusiasm subsided as the dangers of frontal sinus operations became apparent. Logan Turner[9] discussed operative treatment of chronic frontal sinusitis. He said that there were two main principles. In the one, the sinus was preserved and, in the other, the sinus was obliterated. He felt that the Killian operation was the best procedure and gave a higher percentage of cures; however, he stressed the high operative mortality and cited 24 cases of death following operation. Freudenthal[10] also stressed the dangers of surgery. He felt that the majority of cases would improve with intranasal treatment, but that some would need radical operation. He made a large nasofrontal opening. It was not realized until much later that the resulting scar tissue had a strong tendency to contraction.

Barnhill[11] discussed the surgical treatment of empyema of the frontal sinus. He stated that it was necessary to remove completely all pyogenic structures, to provide ample drainage of the operated parts and to get the cavity to heal either by a healthy nonsuppurative lining or by complete obliteration of the space by means of granulation tissue. Of the types of operation he preferred the Coakley type of external obliteration but admitted the great deformity. He took note of the controversy between proponents of conservative and radical treatment. He also emphasized the dangers of both intranasal and external frontal operations. Knapp,[12] in 1908, was the first to approach the frontal sinus externally through the floor; however, if the sinus was large he made a small opening through the anterior wall to reach the upper part of the sinus. Knapp's operation never received widespread recognition until Lynch much later popularized the approach through the floor of the frontal sinus.

In England there was much disagreement about surgery of the frontal sinuses. Many men were dismayed at the deformity and high mortality rate; however, Sir St. Clair Thomson, Watson Williams and others[13] favored the radical operation, either a Killian type or an osteoplastic flap type. Watson Williams was one of the first to mention cicatricial stenosis of the nasofrontal duct after operative enlargement.

In 1912, MacKenty and Cocks[14] discussed the various radical frontal sinus operations. They classified them in three groups: *1.* removal of the anterior wall (Ogston-Luc, Kuhnt), *2.* removal of the floor (Jansen), *3.* removal of both the anterior wall and floor (Riedel, Killian). They preferred Knapp's method of going through the floor and reported six successful cases.

Dissatisfaction with frontal sinus operations has led to the presentation of many modifications of previous procedures. This has continued to the present time. In 1913, McKimmie[15] described a modification of the Killian operation. In the same year McKenzie[16] presented the first comprehensive article in English about diffuse osteomyelitis from nasal sinus suppuration. He said that the osteomyelitis was secondary to infection, either spontaneously or postoperatively, and was more apt to follow acute cases in both categories. In the cases of operated osteomyelitis recorded to that time all patients died. This was a very important step in the realization of operation in the acute phase of disease as a cause of osteomyelitis.

Imperatori[17] reported a case, in 1915, which developed meningitis from an exacerbation of a chronic frontal sinusitis four years after a Killian operation, and died. At autopsy the nasofrontal duct was found to be closed off. This is cited as an instance of the early recognition of the importance of patency of the nasofrontal duct in cases in which the frontal sinus is not completely obliterated. Beck[18] was one of the earlier authors to emphasize the cicatricial closure of the nasofrontal duct after any operation which destroys the mucous membrane and disturbs the bone of the internal nasal crest. He suggested an osteoplastic type of operation which attempted an operation from the frontal sinus to the nose

posterior to the duct itself. This did not seem to be effective. Lothrop[19] then described his new external operation which opened both frontal sinuses and left a large opening into the vault of the nose. This was more successful and is still referred to occasionally. Pattee[20] endorsed the Lothrop procedure, although he felt that the external operation was needed only occasionally. Moore[21] believed that more external frontal operations should be done and preferred Lothrop's operation.

Tilley[22] reported a case of acute osteomyelitis of the frontal bone with operation and recovery. It is mentioned specifically because it is apparently the first case of this sort in which simple external drainage was performed early, and then three weeks later, after partial subsidence of the acute phase, extensive bone removal with obliteration of the frontal sinuses was done. This case was not of the extreme fulminating type. It is interesting to consider this report as a precursor of J. M. Brown's teaching somewhat later.

In 1921, Lynch[23] described his operation, stressing removal of the floor of the frontal sinus, complete ethmoid exenteration and complete removal of the sinus lining. This operation at once gained widespread popularity, and all frontal operations through the floor of the sinus have come to be known as Lynch or Lynch-type operations, even though Knapp and Jansen long antedated Lynch.

Harris,[24] discussing reasons for failure of the Killian operation, mentioned three main difficulties: *1.* failure to obtain complete obliteration, especially behind the orbital arch, *2.* reinfection, *3.* contraction of the nasofrontal duct with trapping in the remains of the sinus. In 1923, Skillern,[25] Fielding Lewis[26] and Howarth[27] wrote articles advocating external operation when needed.

Bulson[28] reported a case of apparently spontaneous osteomyelitis which was cured. The patient refused a radical operation at first, so simple external drainage was done. Two and one-half months later a radical operation was done to remove the diseased bone, long after the acute process subsided. This was similar to Tilley's case.

Sewall,[29] in 1926, first reported performing external operations on the ethmosphenoid-frontal group of sinuses under local anesthesia, the chief advantage being a marked decrease in the amount of bleeding. Hurd[30] reported three unusual cases of frontal sinus disease which demonstrated some of the complications and variations that may occur. The first case required multiple operations several years after having a Killian operation because of the development of several mucoceles. The patient finally recovered after the final operation, which was a modified Lothrop procedure to provide drainage into the nose. The second case developed an ossifying periostitis. This case recovered after an obliterative frontal operation. Infected pockets were found trapped within the ossifying tissue. It has some similarity to one of the cases to be reported in this thesis. The third was a traumatic case in which a Killian-Lothrop type procedure was done. Twenty-five days later the patient needed reoperation. The bony bridge of the orbital ridge, the cribriform plate and the posterior wall of the frontal sinuses were all found to be necrotic. The patient then made a slow but uneventful recovery.

Lillie and Anderson,[31] in 1927, advocated a two-stage operation for the frontal sinuses. They advised preliminary intranasal surgery under local anesthesia as the first stage. In 19 of their 60 cases this was sufficient. In the others they proceeded with the second, or external, stage. They obliterated at least the upper portion of the frontal sinus if the front wall was involved, *e.g.*, with a fistula. They felt that this could be readily done by attention to details such as beveling the edges of the bone; furthermore, this, plus avoidance of a vertical incision, helped to lessen the deformity in their cases. In the same year Gill[32] also advocated obliteration rather than an attempt to restore a badly infected sinus.

Mithoefer[33] was one of the first to advocate grafts to line the nasofrontal duct after a Lynch type operation. He used mucous membrane grafts from the nose and even the lip in some cases. Lothrop,[34] in 1928, stated that there were certain objections to all types of radical operations on the frontal

sinus. He discussed the difficulty of the occurrence of granulations postoperatively; also, the importance of waiting for a quiet stage in which to perform the radical operation.

A good presentation of gross and microscopic pathology in frontal sinus disease was given by Simpson and Harris,[35] in 1928. They found that there were frequently chronic changes in apparently acute cases, and they particularly noted frequent bony involvement such as osteitis, focal necrosis, osteomyelitis and periostitis.

H. W. Lyman[36] reported an unusual case in 1928. His patient had a chronic left frontal sinusitis and was found to have huge frontal sinuses. A Lynch operation was done on the left frontal sinus which required the use of uterine curettes for removal of the lining. The patient was symptom-free for 20 years before a recurrence. His nasofrontal duct was found to be closed on examination, in 1948; an Ingals gold tube was then inserted to be left in permanent position. This use of the Ingals tube for permanent insertion will be discussed in further detail later.

Lewis,[37] in 1929, strongly condemned all radical sinus surgery as destroying all hope of return to anywhere near normal function. In discussing surgical principles in the treatment of chronic sinus disease, Sewall,[38] in regard to the frontal sinus, cited the desirability but the difficulty of obtaining complete obliteration. As an alternative an attempt is made to get drainage. Here one is beset by the difficulties of closure by scar tissue.

Cullom,[39] after reviewing the history of frontal sinus surgery, concluded that Riedel's operation was too deforming and that Killian's operation was ineffective in obliterating the sinus. He advised taking one's time, doing intranasal work first and then proceeding with a Lynch type of operation.

Lillie,[40] in 1931, presented a discussion of postoperative complications following radical external frontal operations. In a series of 158 cases of either Killian or Lynch operations there were complications in 45 cases with three deaths (two from meningitis and one from brain abscess). Among the

nonfatal complications were localized osteomyelitis requiring removal of sequestrums, orbital cellulitis, orbital abscess and erysipelas. Anderson,[41] in a series of 101 cases of either Killian or Lynch operations, stressed two causes of failure: *1.* in the Killian procedure, incomplete operation, resulting in the development of infected pockets; *2.* in the Lynch procedure, contractures of the nasofrontal duct.

In 1933, Mosher with Judd[42] presented his views on treating acute fulminating osteomyelitis of the frontal bone by early radical complete removal of the infected bone with a normal margin, including complete removal of the frontal sinuses.

In the same year Smith[43] described his radical operation in which he exenterated the ethmoids, sphenoids and frontals and then inserted a split thickness skin graft in an attempt to form a new nasofrontal duct. He soon reported[44] a modification of his grafting technique in order to enhance the success of the graft.

Interest in the condition of the lining of the operated frontal sinus was stimulated by the report of Coates and Ersner,[45] in 1930, that in the dog the lining regenerated after its surgical removal; however, as a result of some rather extensive experimental surgery on dogs, Hilding[46] arrived at quite different conclusions. He found that when strips of epithelium were removed they were replaced with heavy scar, which in certain locations interfered with mucus flow, and if near the osteum could block it. If the lining were completely removed, he found[47] that the sinus may obliterate but frequently did not. Some were found to be fairly well restored, but in most of his animals there were scattered cysts filled with mucin in the frontal sinuses. This corroborates the clinical finding of infected pockets found in reoperated cases, a condition which has been reported so many times.

Kleinfeld,[48] in 1934, reported a case of successful collapsing of the frontal sinuses with a Riedel operation. This case had previously had a Killian operation. The obliteration was done because of signs of impending intracranial complications. At the final operation the orbital bridge of bone was found to be partly necrotic. The patient recovered.

Sewall[49] proposed a new type of mucosal flap drawn up into the region of the nasofrontal duct from the lateral nasal wall in an attempt to help form a new duct.

Skillern[50] reported several cases of obliterative frontal sinusitis with infection trapped in the newly growing thick bone. He called this process osteanagenesis. His cases required radical operation with complete removal of the front wall of the frontal sinus.

Diggle and Cawthorne,[51] in 1936, apparently expressing the general trend of thought in England, advocated very conservative treatment of both acute and chronic frontal sinusitis.

In 1936, Reaves[52] demonstrated a practical application of the idea of avoidance of trauma to the nasofrontal duct. In chronic cases, with a fistula, he advised a Killian type of operation leaving a bony bridge, cleaning out the ethmoids into the nose but saving the nasofrontal duct (identified by a probe at operation). He drained his cases externally at first; then the drainage went through the hole in the ethmoids until this scarred up. He felt that by that time the natural frontal duct had recovered its function and could drain the remaining small cavity. This would seem to be a definite advance but was not studied further until some years later.

Adson and Hempstead,[53] in 1937, described an unusual operation for obliteration of the frontal sinus in the presence of osteomyelitis. They used an inverted U-incision, removed the bone from above downward, taking away the posterior wall of the sinus but leaving the anterior wall intact. They tried to remove the mucosa with tincture of iodine. Then the dura and brain came forward into contact with the anterior bony wall to obliterate the sinus. There are several very obvious objections to this procedure, of course. When there is infection in the bone, the anterior wall almost always is involved first. The technical difficulty of removing the sinus membrane would be very great since one of the essential conditions for successful operation is good exposure; furthermore, such wide exposure of the dura left in contact with infected tissue would be highly undesirable. Comments on this procedure have not been offered by any other authors.

In 1937, Mosher[54] said that the frontal sinus operation was still unsatisfactory and that the technique of epidermatizing the enlarged duct or in obliterating the sinus must be perfected. He felt that it would be necessary to remove both anterior and posterior walls to obliterate the frontal sinus successfully. Skillern[55] felt that in cases with osteomyelitic involvement the entire anterior wall of the frontal sinus should be removed.

In 1939, several rather startling ideas were proposed in frontal sinus surgery. None has been generally accepted nor is in use today; however, they are of interest as attempts to solve some of the problems involved. Williams and Fricke[56] proposed the use of radium to keep the nasofrontal opening patent postoperatively but admitted that it was not entirely successful. Matis[57] described a "subperiosteal" operation on the frontal sinus done through the nose and outside the bony edge of the nasal vault. The disadvantage of operating by touch on the frontal sinus seems rather obvious. Kepes[58] proposed a modification of the Killian operation in which he removed the medial half of the bony bridge which Killian originally preserved. Kepes felt that this helped to obliterate the medial portion of the sinus; however, this would make the remaining bone even more prone to necrosis, and photographs accompanying the article show a very marked vertical depression medially. Lothrop[59] insisted that rasping the nasofrontal duct was advisable providing a bony ring was left; however, he then stated that "re-reaming" was easily done subsequently.

Kettel[60] has used a coronal incision from ear to ear, reflecting the scalp downward to expose the frontal bone. Patterson[61] first mentioned the use of chemotherapy in 1939. He felt that prontosil (the first sulfonamide available) made it easier to wait for the best time to operate. He felt that occasionally the anterior wall needed removal, in which case he was careful to bevel the edges of the bone. Jones[62] advocated radical bone removal in cases with osteomyelitis involvement. It is interesting to note that he used the hairline or eyebrow incision and had abandoned the vertical T-incision. Dill[63]

advocated more conservative handling of cases of osteomyelitis with early drainage and later removal of sequestrums and obliteration of sinuses if necessary.

Anthony,[64] in 1940, stated that one of the greatest causes for failure in operation, with recurrence of infection, was closure of the nasofrontal opening. He proposed the permanent insertion of a gold Ingals tube to keep the nasofrontal channel open. He reported 10 cases in which this was successful, although, theoretically, this would not be so desirable as a normal duct lined with ciliated epithelium. This idea certainly seems to be a great advance in a certain type of case in which the sinus is not obliterated and yet the duct cannot be saved. This was done in H. W. Lyman's case upon reoperation, as noted above.

Goodale,[65] in 1942, reviewed 182 cases of frontal sinus disease at the Massachusetts Eye and Ear Infirmary in an effort to determine the most important causes of failure in frontal sinus surgery. In 123 cases of infection there were 38 recurrences, or 30.8 per cent. In 18 cases of mucoceles there were four recurrences, or 22.2 per cent. In the cases of infection, four Killian operations were done with three recurrences; 106 Lynch type operations were done with 30 recurrences; and 13 obliterative operations were done with five recurrences. In the cases of mucocele 11 Lynch type operations were done with three recurrences; and seven obliterative operations were done with no recurrences. He found that the three most important conditions causing recurrence necessitating secondary operation were: *1.* scar tissue, *2.* remnants of frontal sinus floor, *3.* ethmoid extension; therefore, he advised that in either obliterative or drainage operations consideration must be given to complete removal of the frontal sinus floor and the ethmoid cells. In 1942, Fred[66] reviewed 40 cases of fulminating osteomyelitis at the same hospital. He found that lower mortality came with early complete removal of the frontal bone; however, he predicted that the sulfonamides would perhaps convert fulminating cases of osteomyelitis into slowly progressive or localizing types, thus permitting more conservative treatment, *i.e.*, the use of sulfonamides with early drain-

age through the floor and later removal of sequestrums if necessary. Wails[67] advised conservative handling of osteomyelitis following frontal sinusitis. He used early drainage of the sinus followed by removal of sequestrums. He later operated on the sinuses, usually using a Lynch type procedure, occasionally a Riedel type. Jones[68] warned that sulfonamides might mask the clinical course of osteomyelitis and reported a case that died suddenly of a brain abscess. He felt that osteomyelitis of the frontal bone should, therefore, be handled radically as he had previously advocated.

Reporting on a series of surgical experiments on the frontal sinus in dogs, Walsh,[69] in 1943, made some very important observations. He had three groups of animals. In Group I, the nasofrontal duct was enlarged and the rest of the sinus was left untouched. In Group II, the entire sinus was cleaned out and the duct was enlarged. In Group III, the sinus was cleaned out but the nasofrontal duct was left untouched. After the animals were sacrificed, he found that in Groups I and II the sinuses were grossly and microscopically infected. In Group III the sinuses were clean, with completely regenerated lining present. He suggested that there might be a similar response in man as a reason for failures in frontal sinus surgery. He reported three cases of chronic frontal sinus infection in which obliteration was not deemed indicated and in which a Lynch type operation, modified by leaving the nasofrontal duct alone, was done with apparently good results. This is a logical extension of Hilding's work and seems to be an important advance in frontal sinus surgery. Four of the cases discussed in this paper were successfully treated in this manner. The classification of surgical cases into three main categories, of which Walsh's type is one category, will be discussed later.

In 1944, there were four articles published on the use of penicillin in the treatment of osteomyelitis resulting from frontal sinusitis. Iglauer,[70] Kirby and Hepp,[71] Putney[72] and Colbert[73] all agreed that penicillin combined with adequate surgical treatment offered great hopes of reducing the mortality of the disease and permitting more conservative surgical

management. They also found that the sulfonamides were relatively ineffective. Schnitker and McCarthy[74] confirmed these findings in 1945, but emphasized that penicillin is not a substitute for surgery. They found that surgical excision of diseased bone was necessary in most instances and that it could be done as soon as the infection was controlled by penicillin, at which time primary closure of the scalp was a feasible procedure. They then performed a secondary operation for correction of the bony defect three months later, using a tantalum plate.

Discussing causes of failure in the surgical treatment of chronic sinusitis, Russell[75] stated that long standing cases with high grade degeneration of the mucous membrane, denuded bone, necrotic areas and fistulous tracts were unsuitable for drainage and needed complete obliteration of the frontal sinuses. He said that by far the most satisfactory of the obliterative operations was Riedel's, and that although the deformity was severe, corrective plastic surgery was improving. He felt that only a small percentage of cases needed obliteration, but in those that did, one should not temporize. Goodale,[76] in 1945, suggested the permanent placement of a sheet of tantalum foil for the formation of a nasofrontal duct after the Lynch operation. This idea has received considerable favorable attention. Jesberg[77] felt that complete obliteration of the frontal sinus produced too much deformity and proposed an operation in which the lateral half of the sinus was obliterated, converting it into a small cavity which he felt was easier to drain. This is essentially a compromise. Van Alyea[78] strongly advised conservative surgical measures directed toward institution of adequate sinus drainage. He felt that radical removal of sinus mucosa was seldom indicated.

Boise,[79] in 1942, strongly advocated early trephining through the floor of the sinus in acute frontal sinusitis as a measure to prevent the development of complications such as osteomyelitis and intracranial extension. This idea was emphatically seconded by Brown,[80] who also said that penicillin was very useful but did not remove pus already present. After the acute process has subsided, Brown advised a Lynch

type external operation leaving the nasofrontal duct intact according to Walsh's idea. Goodyear[81] also favored early trephining in acute frontal sinusitis. He further recommended the permanent insertion of Ingals' gold tube in all external frontal operations, just as had Anthony a few years before. In attempting to obliterate certain frontal sinuses, Goodyear modified the Killian operation by packing the skin down into what was left of the frontal space. One would expect marked deformity with such a maneuver.

In 1946, Weille[82] reported a new series of cases at the Massachusetts Eye and Ear Infirmary. There were 276 cases, requiring 862 operations. In the Lynch operations closure of the nasofrontal duct was the overwhelming cause of failure. He was impressed by Goodale's tantalum foil and Walsh, Brown and Hilding's idea of leaving the duct alone as promising attempts to remedy this cause of failure. He found that in the obliterative operations, besides the drawback of deformity, frequently the sinus had not actually been obliterated. Mosher[83] summaried his views on frontal sinus surgery in "My Milestones." He reiterated his advice to respect the virginity of the nasofrontal duct because if traumatized it always scars down. He mentioned the work of Hilding and Walsh. He felt that in acute frontal sinusitis early external trephining as advocated by Brown was the best procedure. He discussed the difficulties of the various frontal sinus operations and said he had never been satisfied with any of them. He felt that if the frontal sinus required obliteration, it would be best to remove both anterior and posterior walls. Finally he felt that antibiotics were a tremendous aid in treating osteomyelitis, but that if edema was present and did not regress in 48 hours extensive bone removal should still be practiced.

Negus[84] presented his views on frontal sinus surgery in 1947. In the external operation he used a small incision, did not remove all of the floor, did not curette out the membrane and enlarged the nasofrontal duct, using a skin graft. This is in direct contrast to the opinions found in current American literature. In the same year Erich and New[85] presented

an acrylic obturator which they inserted into the nasofrontal passage a few days postoperatively and left in place for six months. They felt that this gave the passage time to become epithelialized. Besides the obvious objection to wearing a bulky obturator which blocks one side of the nose for six months, there remains the question whether this new passage would not eventually contract and close. Bergara[86] described a very complicated "osteoplastic" operation, making a flap of the anterior frontal sinus wall which he replaced at the completion of the operation. He had nothing to contribute to the problem of dealing with the nasofrontal duct. This type of operation has been reported several times but has never gained acceptance in this country.

In recent years there have been a good many reports of various methods of repair of operative defects in the frontal bone. O'Connor[87] recommended the use of preserved isocartilage grafts. New and Dix[88] used autocartilage grafts from the thoracic cage. Kazanjian and his associates[89,90] have used bone, cartilage, vitallium, tantalum and acrylic. They found that each material had certain advantages and disadvantages, hence they individualized their cases. Even more recently Peer[91] has used diced cartilage grafts and Lierle and Huffman[92] have used autogenous cancellous bone broken up into minute bits. These last two methods are claimed to have the advantage of ease of molding into the desired shape and avoidance of subsequent ridges or bumps.

In January, 1949, Arbuckle[93] reported seven cases of chronic frontal sinusitis with complications, of which six required complete obliteration of one or both frontal sinuses. Winborn[94] advised the following measures in the management of severe frontal sinus infections: heavy doses of penicillin; early trephining of the floor; and later, after subsidence of the acute process, an external operation, usually some type of modification of the Killian, to attempt to obliterate the sinus.

Since this thesis was orginally submitted, Work[95] published an instructive article about the relation of the frontal sinuses to skull injury. He found that in cases in which tantalum plates had been inserted infection was inevitable if the plate

was in contact with the frontal sinus. He also noted that tantalum plates in the forehead were frequently subject to trauma and rather easily became bent. He strongly advised complete obliteration of the frontal sinuses prior to insertion of a tantalum plate for cosmetic repair of frontal defects.

III. SUMMARY OF HISTORICAL REVIEW.

Since the literature is voluminous it is advisable to give a summation. Ogston first opened the frontal sinus through the anterior wall for drainage in 1884. Riedel described complete operative obliteration of the frontal sinus in 1898. Apparently because of the deformity and the risk of the operation it did not become widely used. Killian, in 1903, described his operation and this was immediately greeted with a wave of enthusiasm. The enthusiasm soon abated as the high operative mortality and the frequency of recurrence became apparent. Knapp first entered the frontal sinus through the floor in 1908 and this procedure was popularized, in 1921, by Lynch. It gradually was realized that this operation, too, was prone to lead to recurrence, chiefly because of closure of the nasofrontal opening. It was further gradually realized that radical frontal sinus surgery should not be done in the acute phase of infection except in the presence of a fulminating osteomyelitis. Many attempts have been made to solve the problem of postoperative closure of the nasofrontal duct. Ingals introduced his gold tube for temporary insertion in 1906. It was not until 1940 that Anthony proposed permanent placement of the Ingals tube in the nasofrontal duct region. Goodyear also advised this in 1947. In 1942, Goodale suggested the permanent insertion of a piece of tantalum foil. In 1933, Hilding reported his own experiments showing scarring and closure of the nasofrontal duct. In 1936, Reeves first proposed leaving the nasofrontal duct alone in external frontal sinus surgery. This was followed by Walsh who presented experimental and clinical evidence to further this idea in 1943. Boies, in 1942, and shortly afterward Brown and Goodyear, all strongly advised early trephining of the floor of the frontal sinus in acute infection. This idea has become generally accepted. Many other ideas of variations in operative

technique and procedure have been proposed and discarded. In the last 10 years there have been a good many articles dealing with plastic repair of defects of the frontal bone. In the last few years reports have been presented discussing the use of penicillin in frontal sinus disease and its complications. Penicillin has been found to alter the prognosis of serious cases favorably and allow a more cautious and studied approach to the proper surgical management of difficult cases. It has been found to reduce the incidence of radical operation but the antibiotics are not a substitute for surgery. It has enabled, generally, more conservative management of osteomyelitis. Although most authors seem to have tried to avoid the Riedel obliterative operation, there have been occasional reports through the years of its successful use.

IV. CASE REPORTS.

The following three cases are presented with pertinent comments in each instance. Pictures, X-rays and microphotographs are illustrated. One case is reported from each of the three types of cases. Thirteen of the cases were patients at the Veterans Administration Hospital, Jefferson Barracks, Mo.; four cases are from other hospitals.

Case 1: F. V., a 21-year-old white male entered the Veterans Administration Hospital on the evening of Sept. 5, 1947, on the emergency surgical service and was transferred to otolaryngology the next morning. He gave the history of having had a cold a week previously. Four days before admission he noticed a boil on the back of his neck and severe right frontal headache which prevented sleep. For the past two days he had noticed increasing swelling of the right eye. He also noticed aching in the right upper molar teeth. He had photophobia and nausea. In his past history he had been subject to boils but had never had any headaches. Examination showed a young asthenic man severely and acutely ill. His temperature, on admission, was 101.8° F. There was diffuse edema extending about half way up the forehead. The right upper eyelid was red and very swollen. There was chemosis of the right eye. The nasal mucosa was acutely inflamed and very swollen. There was thick yellow pus in both nasal fossae. There was a sharp deviation of the septum to the right. There was a draining furuncle on the back of the neck. He was started on 50,000 units of penicillin every three hours on admission. The next morning this was increased to 100,000 units every three hours, and he was then started on 1 gm. of sulfadiazine every four hours with an initial dose of 4 gm. The edema of the forehead continued to increase and reached the hairline during the morning after admission. It was felt that he had an acute fulminating right frontal sinusitis with spreading osteomyelitis; therefore, the floor of the right frontal sinus was trephined under local anesthesia at noon on Sept. 6, 1947. The opening

Figs. 1 and 2. Case 1. Front and lateral views showing postoperative appearance following obliteration of the frontal sinuses.

was enlarged with mallet and gouge to a diameter of one-half cm. A small amount of liquid pus was obtained. A flat rubber drain was inserted and a dressing applied. The patient's condition remained stationary for about 24 hours and then began to improve rapidly. The pain, swelling, fever and headache rapidly subsided.

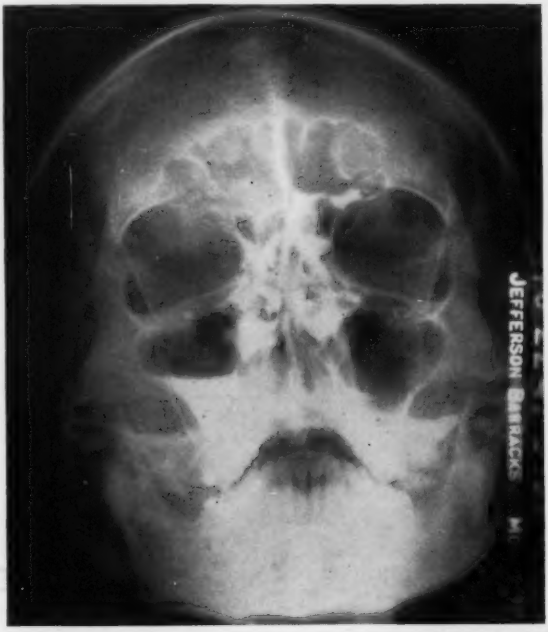

Fig. 3. Case 1. Preoperative X-ray in Waters' position with lipiodol showing extreme thickening of mucosa in frontal sinuses and blocked right nasofrontal duct.

On Sept. 25, 1947, a submucous resection was done to improve the airway and facilitate intranasal drainage. On Oct. 1, 1947, the rubber drain in the right frontal sinus was removed. After the patient's condition had improved, extensive X-ray studies, including lipiodol films, were done to help determine the condition of his sinuses and frontal bone. X-rays showed a pansinusitis on the right side and clouding of the left frontal sinus. The right antrum was clear on irrigation and finally cleared on X-ray. Lipiodol could not be instilled into the right frontal even through a cannula in the nasofrontal duct. Oil instilled in the left frontal sinus showed very thickened irregular lining. The X-rays did not reveal the true extent of bone damage as found at operation. The sulfadiazine was discontinued on Sept. 23, 1947.

On Nov. 6, 1947, a bilateral radical obliterative frontal operation was done under intravenous sodium pentothal anesthesia supplemented with local infiltration with 1 per cent novocaine. A horizontal incision just below the eyebrows was made and the periosteum elevated. A dehiscence, about 1 cm. in diameter, was found in the anterior wall of the left

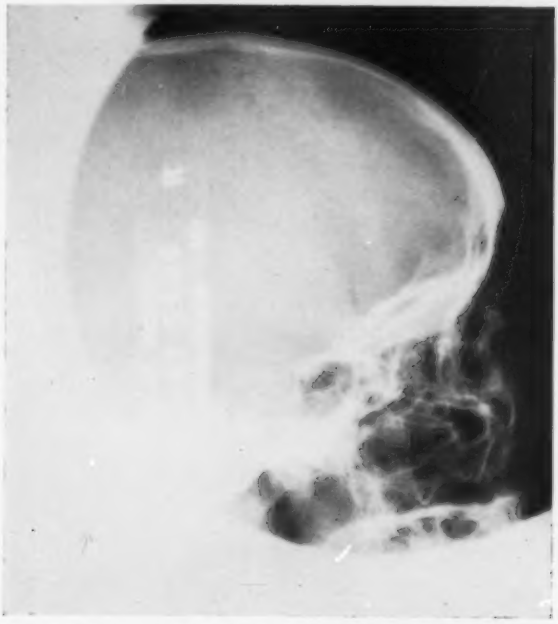

Fig. 4. Case 1. Postoperative X-ray in lateral view showing obliteration of the frontal sinuses and the extent of bone removal.

frontal sinus. On the right side the surgical opening through the floor was uncovered. The anterior wall of both frontals was rough, soft and spongy and was removed completely. The membrane was quite roughened and thickened. This was completely removed. The floor of both frontals was removed and the anterior ethmoids were cleaned out. There was thickened membrane in the ethmoids, but frank pus was found in only two of the left anterior ethmoid cells. The bone at the root of the nose and laterally about the upper outer rim of the orbits was taken down. Rubber drains were placed laterally on each side and brought out at the root of the nose. The periosteum was closed with chromic catgut, the subcutaneous tissue with plain catgut, and the skin with silk. A pressure bandage was applied. Specimens of tissue removed at operation revealed chronic osteomyelitis and chronic sinusitis. His postoperative

course was completely uneventful. The drains were removed on the third postoperative day and the sutures on the fourth and sixth postoperative days. He developed a transient diplopia which rapidly disappeared. Penicillin was stopped on Nov. 17, 1947. The patient left the hospital on leave of absence on Nov. 19, 1947, and returned for periodic checkups until he was given his final discharge on Dec. 20, 1947. He has been completely well since that time.

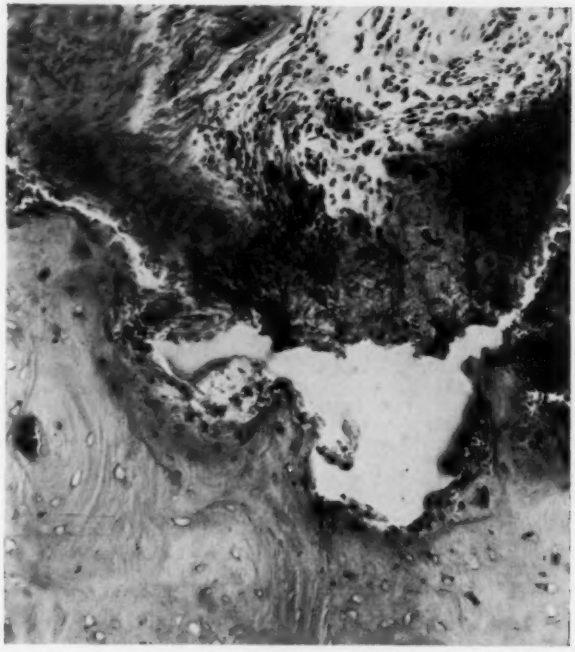

Fig. 5. Case 1. Microphotograph of piece of bone from anterior wall of frontal sinuses showing chronic osteomyelitis.

Comment: This case illustrates the acute fulminating type of infection with early development of osteomyelitis as shown by the spreading edema of the forehead and the extreme toxicity of the patient. Even with heavy doses of penicillin the patient became worse in a matter of hours until an external trephine was done through the floor of the right frontal sinus,

then he rapidly improved. It is important to note the long time elapsing before the obliteration was done. During this time extensive studies, including lipiodol X-ray films, were made in an attempt to determine the condition of the bone and sinuses. The X-rays never did show the full extent of the bone damage. It was finally decided that irreversible changes had occurred in the frontal bone and sinuses and, hence, obliteration was done. The incision below the brow gave excellent exposure, but the resulting scar was found to be more noticeable than a scar within the brow. Beveling the bony edges reduced the bony deformity. The cosmetic result was very good, as the pictures show.

Case 2: C. A. V., a 52-year-old white male, was admitted to McMillan Hospital, St. Louis, Mo., on May 3, 1948, with the following history: This case was reported, originally, by H. W. Lyman, in 1928. He had a chronic suppurative left frontal sinusitis which required a Lynch operation in 1925. The most noteworthy finding was the gigantic size of the frontal sinuses. In 1945, he had an acute flareup with an abscess presenting in the old operative site. This was incised and drained and a gold Ingals tube was inserted in the nasofrontal duct. The tube was spontaneously extruded eight months later. The patient remained symptom-free until April, 1948, when he developed pain and swelling above the left eye. This swelling was incised and drained as an outpatient procedure on May 1, 1948, with immediate relief. On admission to the hospital on May 3, 1948, examination revealed a draining fistula above the medial aspect of the left eye, in the site of the old scar. The right side of the nose was clean. The left side showed hypertrophy of the left inferior turbinate and pus in the upper portion of the nose. The left nasofrontal opening was found to be closed. The diagnosis of acute exacerbation of chronic left frontal sinusitis was made. He was placed on 300,000 units of duracillin daily and, on May 4, 1948, under sodium pentothal anesthesia a gold Ingals tube was inserted for permanent emplacement. This was done by making a short incision under the medial portion of the left eyebrow and rasping downward into the nose. The rasp was then inserted in the nose and manipulated upward until the hole just made from above was entered. The mucosa of the frontal sinus was thickened but relatively intact. A gold Ingals tube, its flaring end compressed by a gelatin capsule, was inserted into the frontal sinus through the nose. The tube was manipulated both from above and below until it was firmly set in position. The wound was closed with silk sutures. Moderate bleeding was controlled by a postnasal pack kept in place during the operation. The sutures were removed on the third postoperative day. He was discharged from the hospital on May 8, 1948, and has remained symptom-free. The tube is still in place.

Comment: This case illustrates the value of permanent insertion of a gold Ingals tube in a case in which the nasofrontal duct had been previously destroyed. The possibility that there were sufficient changes in the sinus lining and even

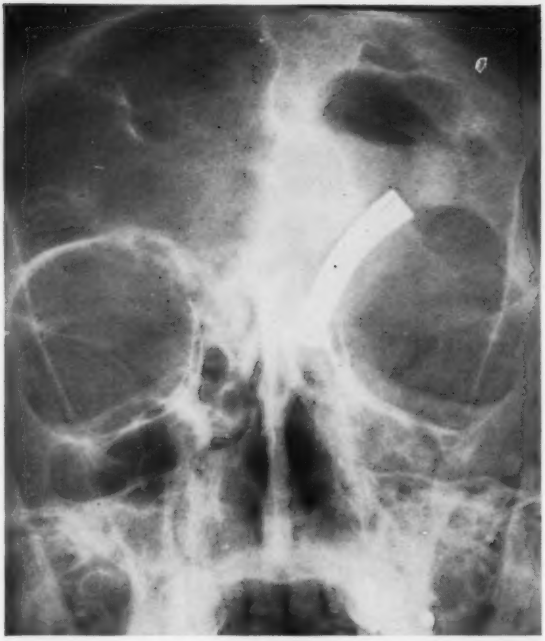

Fig. 6. Case 2. X-ray in front view showing the position of the tube. The extremely large size of the frontal sinuses can be seen, as can the thickening of the sinus membrane.

in the bony walls to warrant obliteration had to be considered; however, the extremely large size of the frontals, combined with the fact that this paient had already had several plastic operations for the repair of the left maxilla, which had been shattered by shrapnel, and the fact that he was symptom-free as long as the nasofrontal duct remained open led to the use of the procedure of permanent insertion of the gold Ingals tube.

Case 3: H. W., a 40-year-old white male, entered the Veterans Administration Hospital on March 25, 1949, with the complaint of right frontal headaches for five years. In 1944, during a search for foci of infection because of backache, an infection of the right maxillary sinus was found and an antrum window was made. He got along fairly well until about

one year before admission when the headaches began to recur. They gradually became worse and he finally came to the hospital. Examination revealed pus in the right middle meatus, streaming over the right inferior turbinate. There was polypoid tissue hanging over the right middle turbinate. The previous antrum window was found to be closed.

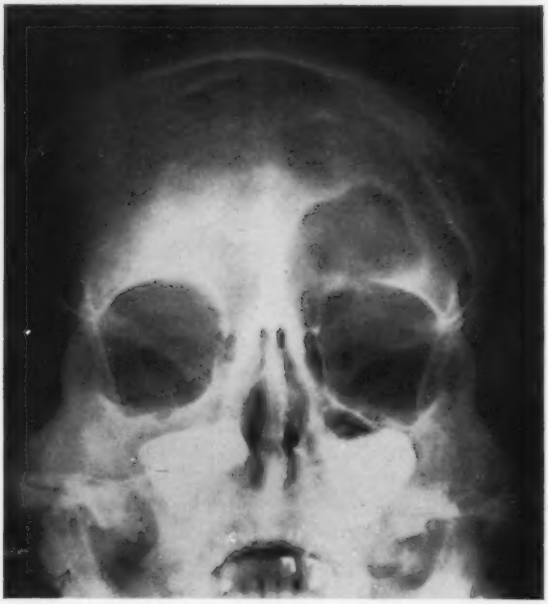

Fig. 7. Case 3. X-ray in Waters' position showing clouding of right frontal sinus with sharply defined area of sclerosis adjacent. Lipiodol in antrums shows thickened lining in the right antrum.

Pus was obtained on antral irrigation. Culture yielded hemophilus influenzae. Sinus X-rays revealed a pansinusitis on the right side with a well defined area of heavy sclerosis about the right frontal sinus. Lipiodol films showed marked thickening of the lining of the right antrum. There was thickened membrane in the right frontal sinus, but the nasofrontal duct was patent.

A right Caldwell-Luc operation, with transantral ethmoidectomy and polypectomy was done on April 4, 1949. The antrum was full of pus and lined with badly infected thickened lining. The lining of the ethmoid cells was thickened with polypoid degeneration. The patient was given 50,000 units of penicillin every three hours from April 5, 1949, to April 8, 1949. His postoperative course was uneventful. Penicillin was resumed on April 20, 1949, and stopped on April 27, 1949. On April 20, 1949, a

right modified Lynch type operation was done under local anesthesia. A gently curved incision was made through the shaved brow on the right side to and through the periosteum. The right frontal sinus was entered through the floor. The bone was smooth, hard and thickened. Pus was found in the sinus and the mucosa was irregular and thickened. There

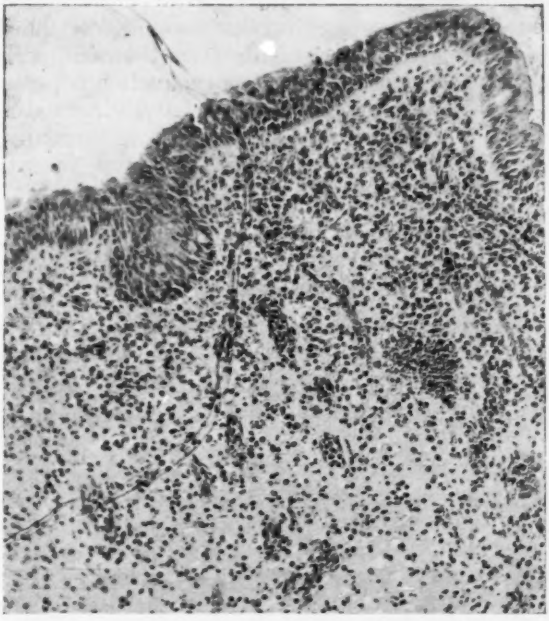

Fig. 8. Case 3. Microphotograph of portion of lining of right frontal sinus showing chronic inflammatory changes in the mucosa.

was a sharp line of demarcation in the mucosa near the entrance to the nasofrontal duct which was patent. The mucosa, at the entrance of the duct, was normal. The mucosa, at the line of demarcation, was divided with a sharp knife and the lining of the sinus completely exenterated, leaving the nasofrontal duct and its entrance intact. There was a small lateral extension of the frontal sinus filled with infected granulations. This was completely exenterated. The floor of the sinus was completely removed. A small rubber drain was placed in the sinus and brought out through the medial end of the incision. The soft tissues were closed with catgut and the skin with silk. A small dressing was applied. The drain was removed on the third postoperative day and the sutures on the fifth. The patient's postoperative course was completely uneventful, and he left the hospital on April 29, 1949, completely relieved of his headaches. He has remain symptom-free since that time.

Comment: This case illustrates the type of sclerosing sinusitis in which the bone surrounding the sinus becomes hypertrophied and is apparently attempting to obliterate the sinus. Certainly there was no evidence of infection in the bone itself and the process may be considered a protective mechanism; however, it is noteworthy that there was a tract filled with infected tissue projecting laterally from the main portion of the right frontal sinus. The sharp demarcation between obviously infected sinus membrane and relatively normal mucosa leading into the nasofrontal duct was also very striking. This was felt to be an ideal case for a Lynch type operation as modified by Walsh and others to leave the nasofrontal duct intact.

V. DISCUSSION.

Consideration of this series of cases shows that cases of frontal sinus infection requiring surgery lend themselves to classification into three categories. The 11 cases in this series in the category requiring obliteration of the frontal sinuses can be classified into three main groups. Group 1 consists of the traumatic cases, in which infection of the frontal bone and sinuses follows external injury. There were two cases of this type. Group 2 includes the acute fulminating frontal sinus infections in which there is rapidly progressive involvement of the bone. There were two cases of this type. Group 3 includes the cases of chronic infection in which there are repeated acute exacerbations and sooner or later the development of infection in the bone as manifested by draining fistulas, necrotic areas in the bone and marked degeneration of the mucous membrane. There were seven cases of this type.

In all of these cases there was severe infection in the frontal sinus itself with irreversible changes in the mucous membrane plus infection in the bony walls of the sinus. Penicillin and other antibiotics, such as streptomycin, are effective aids in treating these cases but do not take the place of surgery. The antibiotics cannot restore a necrotic membrane to normal and are admittedly not as effective in treating infection involving bone as that involving soft tissue alone. In all the

cases in which the frontal sinuses were obliterated, the bone of the front wall was soft and rough, and frequently discolored and necrotic. In contrast, in most of the cases the posterior wall was in good condition. In three of the cases there was a dehiscence in the posterior wall but the rest of the bone of the posterior wall was sound. In one of the traumatic cases almost all of the posterior wall had been destroyed at the time of the accident, or removed at debridement by the neurosurgeon.

In consideration of the proper time for operation, in the acute cases early drainage of the frontal sinuses through the floor is very important. Then a period of antibiotic therapy and observation for subsidence of the acute infection is essential. This should take anywhere from three to six weeks, or longer, depending upon the individual case. If the pathological process is well localized, or is chronic when first seen, the waiting period may not need to be as long.

Clinically, all of the patients whose frontal sinuses were obliterated have done well, with no evidence of reinfection in the frontal region. One case required a long period of observation for possible neurological disease but finally became completely symptom-free. None has shown any evidence that the frontals were not completely obliterated.

Another interesting fact is the presence of severe infection bilaterally in almost every case. In only two cases was one of the sinuses much less involved than the other. In these cases the mucosa was only moderately thickened and the nasofrontal duct was recognizable as such on the left side in contrast to the necrotic tissue in the right side with softening of the anterior wall.

Cosmetically the best results have been obtained by using a horizontal incision gently curved to go through the brow with the connecting portion of the incision over the bridge of the nose at or near the frontonasal suture. The incision is curved downward from the inner end of the brow and then turned at a right angle to go over the bridge of the nose. Special attention is given to beveling the edges of the bone, trimming

down the upper and outer edges of the rim of the orbit, removal of any supraorbital extension of the frontal sinuses posteriorly and exenterating all or part of the ethmoid labyrinths as needed. Gelfoam or oxycel is placed in any dead space which may be found remaining in the central portion of the operative field. Primary closure of the incision is practiced, with long, narrow, flat rubber drains placed to come out at each end of the incision. These are gradually shortened and are removed in a few days. Great care must be taken to approximate the skin edges accurately and smoothly about the bridge of the nose. The periosteum is sutured with interrupted chromic catgut and the skin with interrupted silk sutures. A moderate pressure bandage is kept on the forehead for about one week, being changed as needed. Mechanic's waste is a good material to include in the bandage since it exerts gentle pressure. The skin sutures are removed in four to six days. There is considerable postoperative edema of the upper eyelids but this subsides without difficulty. Transient diplopia has been fairly frequent immediately postoperatively but has not been permanent in any of these cases.

All of the patients were men. In women, the cosmetic aspect requires more attention, but with recent improvements in plastic surgery the main issue of dealing with the infection, which may be fatal if unchecked, should not be sidestepped.

The small amount of constitutional reaction to this major operation is surprising; however, the patient is carefully prepared for surgery. Intravenous fluids, and whole blood if deemed advisable, are used during the operation. Penicillin is used intensively both pre- and postoperatively. Other antibiotics, or one of the sulfonamides, may sometimes be used to supplement the penicillin. Various anesthetics have been used, usually including local infiltration with novocaine and adrenalin plus either sodium pentothal, intravenously, or occasionally nitrous oxide and ether endotracheally.

Admittedly, the cases of frontal sinus infection which need obliteration do not occur very frequently in relation to the total number of cases; however, when the occasion does arise it would seem best to study the patient carefully, prepare him

for operation and then proceed to definitive treatment and not try to temporize. These cases have been presented as illustrations of this viewpoint.

The question arises what to do about other cases in which obliteration is not indicated. It would seem that a certain number of cases would come into the second category, in which there is definite infection in the sinuses, causing disabling symptoms, not relieved by conservative treatment but showing no evidence of extension of infection to the frontal bone itself. There were four of these cases in the series. These cases seem to be well handled by the Lynch procedure as modified by Walsh, Hilding and Reeves to leave the nasofrontal duct intact.

This leaves a third category of cases: those in which the nasofrontal duct has been destroyed, either by disease or by previous operation, and in which obliteration is not indicated or feasible. Case 3 illustrates this type. There was one other similar case, in which the duct had been closed off by a shrapnel wound. Here the insertnon of a gold Ingals tube, to be left in place permanently, seems to be the best solution, as suggested by Anthony and Goodyear; Goodale's tantalum foil is probably an alternative that would be just as effective. Although these cases do not have a duct with ciliary action, apparently gravity assisted by occasional blowing of the nose is amply sufficient to provide drainage and prevent blocking of secretions.

VI. CONCLUSIONS.

1. The literature of frontal sinus surgery is voluminous and controversial.

2. Penicillin is a marvelous aid in treating frontal sinus infections, but is not a substitute for necessary surgery.

3. Cases of frontal sinus infection requiring surgery can be classified in three categories.

4. There are certain cases which still occur that need complete obliteration of the frontal sinuses for cure. These are considered as belonging to the first category.

5. These cases all have extension of infection into the frontal bone with irreversible changes and may be classified in three groups: Group 1, traumatic; Group 2, acute fulminating infections with spreading involvement of bone and impending complications; Group 3, chronic infections with low grade osteomyelitis as exemplified by the presence of chronic draining fistulas, small areas of necrotic bone and extreme degeneration of the mucous membrane. The cases in Group 3 frequently are characterized by repeated acute exacerbations.

6. If obliteration of the frontal sinuses is indicated, the complete obliterative operation should be done. This includes complete removal of the anterior wall and floor of the frontal sinuses and all their contents, but need not include the posterior wall unless it shows definite pathological changes or there is evidence of intracranial extension of the infection. If the ethmoid labyrinths are involved they should be exenterated as well.

7. The cosmetic result following the obliterative operation can be good if careful attention is paid to the incision and to beveling down the bony margins and the upper outer rim of the orbit to avoid a dish-face appearance. The inverted T-incision has been abandoned and a gently curving horizontal incision in the brow is used. The incision is closed primarily with careful attention to the proper approximation of tissues, especially about the root of the nose. Long, narrow drains are placed at the outer ends of the wound. A moderate pressure bandage is applied. If the patient feels that he wants a plastic operation later, recent developments in plastic surgery offer several methods of correcting the bony defect.

8. Certain other cases of frontal sinusitis in which the bone is not involved are well treated by the Lynch operation as modified to leave the nasofrontal duct intact. These are considered as belonging to the second category.

9. There are a few cases coming to surgery which are not covered by either of the above categories. These include cases in which the nasofrontal duct has been destroyed by disease or previous operation but in which obliteration is not indi-

cated. These cases are best treated by the permanent insertion of the gold Ingals tube. These are considered as belonging to the third category.

10. The classification of cases of frontal sinus infection requiring surgery into these three categories for consideration of the proper surgical approach appears logical and is based on the pathological conditions present and on the most rational method of correcting the condition in each type of case.

VII. SUMMARY.

The literature of frontal sinus surgery is reviewed. Three case reports are presented. The first illustrates obliteration of the frontal sinus, performed in 11 cases. The second illustrates permanent insertion of the gold Ingals tube, performed in two cases. The third illustrates preservation of the frontal sinus with an intact nasofrontal duct, performed in four cases. Various aspects of the operations are discussed and certain conclusions drawn.

REFERENCES.

1. Ogston, A.: Trephining the Frontal Sinuses for Catarrhal Disease. *Med. Chron.*, Manchester, 1:235-238, 1884.

2. Riedel: Dissertation, Jena, 1898, as quoted by Cullom, M. M.: External Operation on the Frontal Sinus. *Arch. Otolaryngol.*, 11:304-321, Mar., 1930.

3. Killian, G.: Die Killiansche Radical Operation Chronischer Stirnhohleneiterungen. *Arch. f. Laryngol. u. Rhinol.*, 13:59-88, Berlin, 1903.

4. Coffin, L. A.: The Choice of Operation for the Relief of Chronic Empyema of the Frontal Sinus and the Anterior Ethmoid Cells. *Tran. Amer. Laryngol. Assn.*, 27:116-125, 1905.

5. Richards, G. L.: My Personal Experiences with Empyemata of the Frontal Sinus. *Tran. Amer. Laryngol. Assn.*, 27:125-148, 1905.

6. Coakley, C. G. (and others): Symposium: Frontal Sinusitis — Diagnosis, Treatment and Results. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, 11:101-182, 1905.

7. Casselberry, W. E.: The Frontal Sinus. *Tran. Amer. Laryngol. Assn.*, 27:227-230, 1905.

8. Ingals, E. Fletcher: New Operation and Instruments for Draining the Frontal Sinus. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, 11:183-189, 1905.

9. Turner, A. Logan: The Operative Treatment of Chronic Suppuration of the Frontal Sinus. *Jour. A. M. A.*, 44:346-355, Feb. 4, 1905.

10. Freudenthal, W.: The Radical Operation for Empyema of the Frontal Sinus. *Jour. A. M. A.*, 44:446-455, Feb. 11, 1905.

11. Barnhill, J. F.: Some Surgical Principles Essential to the Cure of Frontal Sinus Empyemas. *Ann. Otol., Rhinol. and Laryngol.*, 17:736-745, Sept., 1908.

12. Knapp, A.: The Surgical Treatment of Orbital Complications in Diseases of the Nasal Accessory Sinuses. *Jour. A. M. A.*, 51:299-301, July 25, 1908.

13. Thomson, St. Clair; Moore, Irwin, and Low, Stuart: Cases Illustrating the Surgical Treatment of Frontal Sinus Suppuration; and Watson-Williams, P.: The Present Position of the Treatment of Purulent Discharge from the Frontal Sinuses. *Proc. Roy. Soc. Med.*, Vol. 4, Session 1910-1911, Part II, Laryngol. Sec., pp. 123-127, 1911.

14. MacKenty, J. E., and Cocks, G. H.: The Radical Frontal Sinus Operations. *Manhattan Eye and Ear Hosp. Rep.*, 13:78-93, New York, 1912.

15. McKimmie, O. A.: The Requirements of an Ideal Operation for Chronic Frontal Sinusitis. *Tran. Amer. Laryngol. Assn.*, 35:299-305, 1913.

16. McKenzie, D.: Diffuse Osteomyelitis from Nasal Sinus Suppuration. *Jour. Laryngol. and Otol.*, 28:6, 79 and 129, 1913.

17. Imperatori, C. J.: Report of a Case of Chronic Frontal Sinusitis, Sphenoiditis, Influenzal Meningitis; Death; Necropsy, The Laryngoscope, 25:580-583, 1915.

18. Beck, J. C.: External Frontal Sinus Operation. *Jour. A. M. A.*, 67: 1811-1815, Dec. 16, 1916.

19. Lothrop, H. A.: The Treatment of Frontal Sinus Suppuration. The Laryngoscope, 27:1-13, Jan., 1917.

20. Pattee, J. J.: The Lothrop Operation for Frontal Sinusitis. *Ann. Otol., Rhinol. and Laryngol.*, 27:677-683, June, 1918.

21. Moore, T. W.: Some Observations on Diseases of the Frontal Sinuses. *Ann. Otol., Rhinol. and Laryngol.*, 29:453-454, June, 1920.

22. Tilley, H.: Acute Osteomyelitis of the Frontal Bone: Operation: Recovery. *Brit. Med. Jour.*, Vol. II, 1917, pp. 7-9, July 7, 1917.

23. Lynch, R. C.: The Technique of a Radical Frontal Sinus Operation Which Has Given Me the Best Results. The Laryngoscope, 31:1-5, Jan., 1921.

24. Harris, T. J.: Causes of Failure in the Radical Operation on the Frontal Sinus. *Jour. A. M. A.*, 77:1147-1151, Oct. 8, 1921.

25. Skillern, R. H.: The End-Results of Radical Operations on the Accessory Sinuses. *Ann. Otol., Rhinol. and Laryngol.*, 32:139-148, Mar., 1923.

26. Lewis, Fielding O.: The Radical Frontal Sinus Operation, with Report of Cases. *Ann. Otol., Rhinol. and Laryngol.*, 32:305-315, Mar., 1923.

27. Howarth, W. G.: A Radical Frontal Sinus Operation. *Jour. Laryngol. and Otol.*, 38:341-343, July, 1923.

28. Bulson, A. E., Jr.: Osteomyelitis of the Frontal Bone as a Complication of Frontal Sinusitis. *Jour. A. M. A.*, 86:1:246-250, Jan. 23, 1926.

29. Sewall, E. C.: External Operation on the Ethmosphenoid-Frontal Group of Sinuses Under Local Anesthesia. *Arch. Otolaryngol.*, 4:377-411, Nov., 1926.

30. Hurd, L. M.: Frontal Sinus Disease—Three Unusual Consecutive Cases. The Laryngoscope, 36:894-897, Dec., 1926.

31. Lillie, H. I., and Anderson, C. M.: The Two-Stage Operation for Suppurative Frontal Sinusitis with External Manifestations. *Arch. Otolaryngol.*, 5:152-161, Feb., 1927.

32. Gill, E. G.: Chronic Frontal Sinusitis: Surgical Treatment. *South. Med. Jour.*, 20:612-616, Aug., 1927.

33. MITHOEFER, W.: External Operation on the Frontal Sinus. A Critical Review. *Arch. Otolaryngol.*, 7:133-149, Feb., 1928.

34. LOTHROP, O. A.: The Management of Frontal Sinus Infection. *N. Eng. Jour. Med.*, 199:1078-1081, Nov. 29, 1928.

35. SIMPSON, W. L., and HARRIS, R.: Gross and Microscopic Pathology in 23 Consecutive External Frontal Ethmosphenoid Operations. *Ann. Otol., Rhinol. and Laryngol.*, 37:452-476, June, 1928.

36. LYMAN, H. W.: Gigantic Frontal Sinus Requiring External Operation. *Ann. Otol., Rhinol. and Laryngol.*, 37:1195-1200, Dec., 1928.

37. LEWIS, E. R.: Considerations For and Against Curetting and Exenterating Sinus Operations. *Ann. Otol., Rhinol. and Laryngol.*, 38:685-690, Sept., 1929.

38. SEWALL, E. C.: General Surgical Principles Employed in the Treatment of Chronic Disease of the Nasal Sinuses. *Ann. Otol., Rhinol. and Laryngol.*, 38:995-1004, Dec., 1929.

39. CULLOM, M. M.: External Operation on the Frontal Sinus. *Arch. Otolaryngol.*, 11:304-321, Mar., 1930.

40. LILLIE, H. I.: Postoperative Complications of the Radical External Frontal Sinus Operations. *Tran. Amer. Laryngol. Assn.*, 53:136-145, 1931.

41. ANDERSON, C. M.: External Operation on the Frontal Sinus. Causes of Failure. *Arch. Otolaryngol.*, 15:739-745, May, 1932.

42. MOSHER, H. P., and JUDD, D. K.: An Analysis of Seven Cases of Osteomyelitis of the Frontal Bone, Complicating Frontal Sinusitis. THE LARYNGOSCOPE, 43:153-212, Mar., 1933.

43. SMITH, F.: Chronic Sinus Disease. Its Present Status. *Jour. A. M. A.*, 100:402-407, Feb. 11, 1933.

44. SMITH, F.: Additions to the Complete Operation on the Frontoethmosphenoid Sinuses. *Arch. Otolaryngol.*, 23:184-186, Aug., 1935.

45. COATES, G. M., and ERSNER, M. S.: Regeneration of the Mucous Membrane of the Frontal Sinus After Its Surgical Removal (in the Dog). *Arch. Otolaryngol.*, 12:642-648, Nov., 1930.

46. HILDING, A.: Experimental Surgery of the Nose and Sinuses. Results Following Partial Removal of Lining Mucous Membrane from the Frontal Sinus of the Dog. *Arch. Otolaryngol.*, 17:321-327, Mar., 1933.

47. HILDING, A.: Experimental Surgery of the Nose and Sinuses. Results Following Complete Removal of Lining Mucous Membrane from the Frontal Sinus of the Dog. *Arch. Otolaryngol.*, 17:760-768, June, 1933.

48. KLEINFELD, L.: Frontal Sinusitis — Collapse Operation. *Jour. Mt. Sinai Hosp.*, I:20-22, May-June, 1934.

49. SEWALL, E. C.: External Frontoethmosphenoid Operation — New Mucosal Flap. *Arch. Otolaryngol.*, 20:57-60, July, 1934.

50. SKILLERN, S. R.: Obliterative Frontal Sinus. *Arch. Otolaryngol.*, 23:267-284, Mar., 1936.

51. DIGGLE, F. H., and CAWTHORNE, T.: The Treatment of Acute Frontal Sinusitis. *Jour. Laryngol. and Otol.*, 51:318-328, May, 1936.

52. REEVES, E.: External Radical Frontal Sinus Operation with Retention of the Original Osteum Frontale. THE LARYNGOSCOPE, 46:446-459, June, 1936.

53. ADSON, A. W., and HEMPSTEAD, B. E.: Osteomyelitis of the Frontal Bone Resulting from Extension of Suppuration of the Frontal Sinus. *Arch. Otolaryngol.*, 25:363-372, Apr., 1937.

54. MOSHER, H. P.: Unsolved Problems in Otolaryngology. *Tran. Amer. Acad. Ophthal. and Otolaryngol.*, 42:27-47, 1937.

55. Skillern, S. R.: Osteomyelitic Invasion of the Frontal Bone Following Frontal Sinus Disease. *Ann. Otol., Rhinol. and Laryngol.*, 48:392-411, June, 1939.

56. Williams, H. L., and Fricke, R. E.: Use of Radium in Maintaining Patent Frontonasal Opening in External Operations on Frontoethmoid Group of Sinuses—Preliminary Report. *Ann. Otol., Rhinol. and Laryngol.*, 48:412-418, June, 1939.

57. Matis, J. E.: The Principles of Modern Frontoethmoidal Operations and the Subperiosteal Method. *Jour. Laryngol. and Otol.*, 54:649-662, Nov., 1939.

58. Kepes, P.: Modifikation der Killian'schen radicalen Stirnhohlen-Operation. *Acta Otolaryngol.*, 30:110-118, Mar.-Apr., 1942.

59. Lothrop, O. A.: Evaluation of the Lothrop Operation. *Arch. Otolaryngol.*, 33:72-77, Jan., 1941.

60. Kettel, K.: Osteomyelitis of the Frontal Bone. Surgical Treatment: Which Way of Approach Is the Best? *Arch. Otolaryngol.*, 31:622-625, Apr., 1940.

61. Patterson, N.: External Operation on Frontal and Ethmoid Sinuses. *Jour. Laryngol. and Otol.*, 54:235-244, May, 1939.

62. Jones, A. C.: Osteomyelitis of the Frontal Bone with Report of 13 Cases. *Ann. Otol., Rhinol. and Laryngol.*, 49:713-727, Sept., 1940.

63. Dill, J. L.: Osteomyelitis of the Frontal Bone. The Laryngoscope, 52:267-274, Apr., 1942.

64. Anthony, D. H.: Use of Ingals' Gold Tube in Frontal Sinus Operations. *South. Med. Jour.*, 33:949-955, Sept., 1940.

65. Goodale, R. L.: Some Causes of Failure in Frontal Sinus Surgery. *Ann. Otol., Rhinol. and Laryngol.*, 51:648-652, Sept., 1942.

66. Fred, G. B.: Acute Fulminating Osteomyelitis of the Frontal Bone. A Review and a Prediction. *Ann. Otol., Rhinol. and Laryngol.*, 51:638-647, Sept., 1942.

67. Wails, T. G.: The Practical Handling of Osteomyelitis Following Frontal Sinusitis. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, 48:252-259, 1942.

68. Jones, A. C.: Incorrect Treatment of Osteomyelitis of the Frontal Bone. *Arch. Otolaryngol.*, 38:547-549, Dec., 1943.

69. Walsh, T. E.: Experimental Surgery of the Frontal Sinus. The Rôle of the Ostium and Nasofrontal Duct in Postoperative Healing. The Laryngoscope, 53:75-92, Feb., 1943.

70. Iglauer, S.: Two Cases of Osteomyelitis Treated with Penicillin. The Laryngoscope, 54:150-156, Jan., 1944.

71. Kirby, W. M., and Hepp, V. E.: Treatment of Osteomyelitis of the Facial Bones with Penicillin. *Jour. A. M. A.*, 125:1019-1022, Aug. 22, 1944.

72. Putney, F. J.: Uses of Penicillin in Diseases of the Nose and Throat. *Jour. A. M. A.*, 126:620-622, Nov. 4, 1944.

73. Colbert, R. M.: Osteomyelitis of the Frontal Bone Treated with Penicillin. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc., Inc.*, 49:165-171, 1944.

74. Schnitker, M. T., and McCarthy, W. D.: Osteomyelitis of the Skull—Its Treatment with Penicillin and Repair of the Defect with Tantalum. *Surg.*, 18:94-115, July, 1945.

75. Russell, H. G.: Causes of Failure in Surgical Treatment of Chronic Sinusitis. *Jour. Laryngol. and Otol.*, 60:31-39, Jan., 1945.

76. Goodale, R. L.: The Use of Tantalum in Radical Frontal Sinus Surgery. *Ann. Otol., Rhinol. and Laryngol.*, 54:757-762, Dec., 1945.

77. JESBERG, S.: Surgical Treatment of Chronic Frontal Sinusitis. *Arch. Otolaryngol.*, 43:66-69, Jan., 1946.

78. VAN ALYEA, O. E.: Frontal Sinus Drainage. *Ann. Otol., Rhinol. and Laryngol.*, 55:267-277, June, 1946.

79. BOIES, L. R.: Acute Frontal Sinusitis: The Trephine Operation for Drainage in Selected Cases. THE LARYNGOSCOPE, 52:458-468, June, 1942.

80. BROWN, J. M.: Frontal Sinusitis. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, 50:314-317, 1946.

81. GOODYEAR, H. M.: Surgery of the Frontal Sinus. THE LARYNGOSCOPE, 57:340-353, May, 1947.

82. WEILLE, F. G.: The Problem of Secondary Sinus Surgery. *Tran. Amer. Laryngol. Assn.*, 67:151-188, 1946.

83. MOSHER, H. P.: My Milestones. Part I. THE LARYNGOSCOPE, 56:639-652, Nov., 1946.

84. NEGUS, V. E.: The Surgical Treatment of Chronic Frontal Sinusitis. *Brit. Med. Jour.*, I:135-136, Jan. 25, 1947.

85. ERICH, J. B., and NEW, G. B.: An Acrylic Obturator Employed in the Repair of an Obstructed Frontonasal Duct. *Tran. Amer. Acad. Ophth. and Otolaryngol.*, 51:628-632, July-Aug., 1947.

86. BERGARA, R.: Osteoplastic Operation on the Large Frontal Sinus in Chronic Suppurative Sinusitis. End-Results. *Tran. Amer. Acad. Ophth. and Otolaryngol.*, 51:643-647, July-Aug., 1947.

87. O'CONNOR, G. B.: Contour Reconstruction After External Frontal Sinus Operation. *Ann. Otol., Rhinol. and Laryngol.*, 47:183-188, Mar., 1938.

88. NEW, G. B., and DIX, C. R.: The Repair of Defects of the Frontal Bone. *Surg., Gynec. and Obst.*, 70:698-701, Mar., 1940.

89. KAZANJIAN, V. H., and CONVERSE, J. M.: Reconstruction After Radical Operation for Osteomyelitis of the Frontal Bone. *Arch. Otolaryngol.*, 31:94-112, Jan., 1940.

90. KAZANJIAN, V. H., and HOLMES, E. M.: Reconstruction After Radical Operation for Osteomyelitis of the Frontal Bone. *Surg., Gynec. and Obst.*, 79:397-411, Oct., 1944.

91. PEER, L. A.: Diced Cartilage Grafts. *Arch. Otolaryngol.*, 38:156-165, Aug., 1943.

92. LIERLE, D. M., and HUFFMAN, W. C.: A Simplified Method of Obliterating Frontal Bone Defects. THE LARYNGOSCOPE, 59:61-65, Jan., 1949.

93. ARBUCKLE, M. F.: Treatment of Infection of the Frontal Sinuses and Its Complications. *Ill. Med. Jour.*, 95:27-32, Jan., 1949.

94. WINBORN, C. D.: Frontal Sinus Infections—Complications and Management. *Ann. Otol., Rhinol. and Laryngol.*, 58:280-288, Mar., 1949.

95. WORK, W. P.: Parasanal Sinuses in Relation to Skull Injury. *Jour. A. M. A.*, 141:977-981, Dec. 3, 1949.

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THE MANAGEMENT OF TINNITUS.*†

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I. INTRODUCTION:

A reorientation is necessary in our attitude toward the management of tinnitus. Tinnitus is a symptom, not a disease. It is, therefore, unscientific to speak of the treatment of a symptom of almost any otologic disease. There can be no proper discussion of the treatment of tinnitus as a pathologic entity. It is unreasonable to expect any one method of treatment, drug or operation, to favorably influence a symptom which may arise from a host of causes in a number of different locations in the auditory pathway.

The patient who suffers from tinnitus, however, deserves much of our thought. The management of the problem presented in a given patient is a sober challenge.

II. TERMINOLOGY AND DEFINITIONS:

The terminology employed in the discussions concerning tinnitus has been very confusing and has resulted in much of the lack of clarity on this subject.

The word tinnitus, derived from the Latin, refers to a jingling or tinkling. It is necessary immediately to distinguish two separate applications of the term tinnitus, namely, tinnitus aurium and tinnitus cerebri (tinnitus cranii). The former term refers to tinnitus of ear origin and the latter to tinnitus of intracranial origin. The two types of tinnitus are

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entirely different in location, but they may co-exist. Accordingly, the first hurdle in the terminology is the distinction between these two basic types of sensory phenomena.

Tinnitus aurium is usually localized to the one ear or both ears, and described by the patient in some specific manner, *i.e.*, steam escaping, seashell, ocean roar, bells ringing, whistles, click, rumble, static, etc.

Tinnitus cerebri is not too well localized by the patient. It is in the head and usually is described as a generalized roaring. It is rarely a specific type of noise. It is diffuse and may even be attributed to a roaring in the neck and occasionally thorax. It is usually unaccompanied but may occasionally be accompanied by tinnitus aurium. It properly belongs in the domain of the general physician or neurologist. It may be due to vascular intracerebral disease and usually has same psychosomatic components. Occasionally it is difficult to distinguish between tinnitus cerebri and tinnitus aurium.

Tinnitus aurium must be divided into two distinct categories, which may be defined as follows:

1. True or subjective tinnitus is the subjective cortical perception of auditory sensations inaudible to anyone but the

TABLE A.

TINNITUS	
STATIC	DYNAMIC
NONVIBRATORY	VIBRATORY
TRUE	PSEUDO
SUBJECTIVE	OBJECTIVE
INTRINSIC	EXTRINSIC
COMPENSATED	
UNCOMPENSATED	

patient. Such sensations may be due to an actual subaudible somatic source, or as is most generally the case, to an auditory paresthesia from anywhere in the auditory pathway.

2. Pseudo or objective tinnitus is the subjective cortical perception of auditory sensations potentially audible to an examiner, as well as to the patient.

These two subtypes have been given several synonyms, which are summarized in Table A.

III. PSEUDO OR OBJECTIVE TINNITUS:

This type of tinnitus has been characterized by Fowler as "vibratory." Numerous cases have been reported in the literature, but in general, this type of tinnitus is relatively *very uncommon* as compared to the extremely common subjective or true tinnitus. Pseudo or objective tinnitus is usually either vascular or muscular in origin.

The vascular phenomena responsible for pseudo or objective tinnitus are usually connected with organic vascular disease, most frequently characterized in the intracranial aneurysm. This type produces an auditory murmur which is quite audible to an examiner.

Muscular causes for pseudo or objective tinnitus will usually arise from either the tensor tympani, stapedius or salpingopharyngeus muscles. These muscular clicks are easily audible by an examiner. Frequently the audible click may be accompanied by a visible phenomenon in the tympanum or in the tubal area. The mechanism underlying these unusual muscular contractions is not well understood. It has been presumed that psychogenic factors of the "tic mechanism" variety play a great part in the origin of the muscular variety of objective tinnitus. Fatigue factors and involuntary reflex muscular contractions may play a part in the causation.

IV. TRUE SUBJECTIVE TINNITUS — DIAGNOSTIC ASPECTS:

The patient with true subjective tinnitus presents a double diagnostic problem. The first aspect of the problem is the

etiologic diagnosis, and the second aspect is the psychosomatic diagnosis. A valid assessment of any patient presenting the symptom of true or subjective tinnitus demands that the diagnosis be dual; namely, etiologic and psychosomatic.

1. Etiologic Diagnosis:

The etiologic diagnosis must be based upon *a.* anatomic localization and *b.* pathologic physiology. As illustrated in Table B, the anatomic location may include any portion of the auditory pathway from the external auditory canal to the auditory cortex. The pathologic lesion may be of almost any type of derangement peculiar to the anatomic site. A brief

TABLE B.

ETIOLOGY OF TINNITUS (DIFFERENTIAL DIAGNOSIS).

ANATOMIC LOCATION	PATHOLOGIC LESION
EXT. AUD. CANAL	Anemia
TYMPANUM	Hyperemia
a. Memb. tymp.	Edema
b. Tymp. muscles	Serous exudate
c. Ossicles	Mucus exudate
d. Tymp. plexus	Purulent exudate
e. Tymp. vessels	Hemorrhage
f. Eust. tube	Inflammation
g. Fenestrae—Rotund	Allergy
Ovale	Neuronitis
COCHLEA	Necrosis
a. Perilymph	Fibrosis
b. Endolymph	Tumor
c. Organ of Corti	Aneurysm
VIIIth NERVE	Capillary fragility
a. Spiral ganglia	Stasis
b. Trunk	Sludging
INTRACEREBRAL	Vascular spasm
a. Ventral and dorsal nuclei	Vascular sclerosis
b. 2nd order neurons	Vasomotor paralysis
c. Med. gen. body	
d. 3rd order neurons	
e. Cortex	

listing of anatomic locations and pathologic lesions as shown in Table B illustrates the great complexity of possibilities to be considered under etiologic diagnosis. In addition to the infinite variety of lesions and locations, there are extensive variations in intensity of pathological disturbances and extent of anatomical involvement.

The most common anatomic location is probably in some portion of the organ of Corti and its neuronal extensions. The most common pathologic lesion is probably vascular.

It must be remembered in connection with the etiologic diagnosis that tinnitus is one of the five cardinal symptoms of ear disease, the other four being otalgia, otorrhea, vertigo and deafness.

2. Psychosomatic Diagnosis:

The psychosomatic diagnosis or perhaps, more logically, the "psychosomatic loading factor" is of as great importance in the proper analysis of a patient with tinnitus as the etiologic diagnosis. The psychosomatic diagnosis must be approached from two standpoints: *a.* the emotional threshold or sensitivity level of the patient, and *b.* specific psychodynamic problems. Dr. E. P. Fowler, Sr., should be greatly commended for drawing our attention to this approach and for emphasizing its great practical importance in the management of the patient with tinnitus (see Table C).

TABLE C.

TINNITUS IS A DOUBLE DIAGNOSTIC PROBLEM.	
ETIOLOGY—DIFFERENTIAL DIAGNOSIS	PSYCHOSOMATIC WEIGHT (LOADING FACTOR)
a. ANATOMIC LOCATION	*a.* EMOTIONAL THRESHOLD (sensitivity level)
b. PATHOLOGIC PHYSIOLOGY	*b.* SPECIFIC PHOBIAS (anxiety states, conversion mechanisms)

A. Emotional Threshold—Sensitivity Level of Patient:

There is no correlation between the actual intensity of tinnitus as measured audiometrically and the amount of suffering of the patient. It is important, therefore, to attempt to assess the excitability level of the patient. This is not a simple matter and requires keen clinical judgment.

B. Specific Psychodynamic Problems:

The specific psychodynamic problems that must be considered are primarily in the nature of phobias, anxiety states and conversion mechanisms. Some of the most common problems are those involving the fear of impending insanity, the fear of a brain tumor, and the fear of total deafness, particularly if the patient already has a significant hearing loss.

V. TINNITUS ANALYSIS—RECOMMENDED PROCEDURE:

1. History:

A chronological detailed history is of prime importance and need not be stressed further.

2. Otologic Examination:

The otologic examination should include, in addition to a thorough physical examination of the ears, nose and throat, pure tone and speech audiograms, recruitment tests, tinnitus localization on the audiometer, both as to intensity and frequency, and caloric vestibular tests.

3. Simple Medical Findings including blood pressure, fundus examination, hemoglobin determination and a rough neurological screening are indispensable. Serologic and Roentgen examinations may be necessary.

4. Guarded, Indirect Psychosomatic Inventory:

This is last but not least in the investigation. Such an inventory is distinctly the responsibility of the otologist and should not be shifted to the psychiatrist unless major psychi-

atric problems are uncovered and demand major psychiatric help. In this inventory it is important not to suggest negative facts, such as insanity, deafness, tumor, etc. It is important to allow the patient to tell his own story and mention his own specific anxiety states and fears, without prompting.

Such an approach to the patient with tinnitus will usually yield a reasonably accurate etiologic diagnosis and will probably establish the proper psychosomatic weight carried by this particular patient-tinnitus combination.

VI. MANAGEMENT OF THE PATIENT WITH TINNITUS:

The goal in the management of the patient with tinnitus is, therefore, a dual one. The first goal in management is, of course, specific therapy wherever and whenever possible. The second goal is the attempt to change an uncompensated tinnitus into a compensated tinnitus in order to make the tinnitus an acceptable sensory phenomenon and detract from its psychosomatic load on the patient.

A. Specific Therapy:

The specific therapy to be applied in the patient with tinnitus depends entirely, of course, upon the anatomic location and the pathologic change. Inasmuch as this aspect entails the entire scope of otologic diseases, it is not within the sphere of the present discussion to enter upon this subject even lightly. We will all agree that in many instances specific therapy is known and successful. We will as easily and quickly agree that in many instances specific therapy is either unknown or impossible at the present time. Suffice it to say that there are numerous instances where specific therapy is applicable. It can be shown, for example, that tinnitus due to serous tympanic effusions may be quickly relieved by appropriate treatment. Tinnitus due to toxic neuropathies produced by drugs may in some instances be relieved by withdrawal of the offending drugs. Tinnitus due to otosclerosis is occasionally improved following fenestration surgery and occasionally improved with a hearing aid. Tinnitus due to acute

acoustic trauma is usually relieved by the removal of the offending noise source. There are numerous other illustrations of the reversibility of the symptom of tinnitus. Time does not allow complete discussion.

B. Psychotherapy:

In most instances simple psychotherapy by the otologist is far more effective than such therapy in the hands of the psychiatrist. Occasionally such therapy in the hands of the psychiatrist might actually be detrimental where the problem is not a major psychiatric disturbance. It is only in rare instances that expert psychiatric help should be employed in the patient with tinnitus.

It is of the utmost importance to determine why the patient sought otologic advice at this particlar time.

a. Was it because of the actual intensity of the tinnitus? This is a very rare cause.

b. Was it because of anxiety regarding the meaning of this tinnitus and regarding its possible future repercussions? This is a very common cause for a patient seeking otologic advice.

1. Technique: A thorough examination is obviously important for two reasons: *a.* for actual diagnostic necessity, and *b.* to convince the patient of the thoroughness and validity of the otologist's reassurance.

2. It is necessary to explain to the patient what tinnitus represents, namely, that it is a real and not an imagined thing, that it is not an *illusion nor a hallucination or delusion. It is an auditory paresthesia.* In this regard it is frequently valuable to give the patient the example of the phantom limb with neuron memory and to dismiss from the patient's mind the mistaken and concealed fear that the tinnitus is a sign of mental deterioration.

3. Be sure to explain to the patient *"what tinnitus is not."* Reassure all his doubts, guardedly but firmly.

4. Be optimistic regarding the duration and the severity of tinnitus, remembering that in most cases of tinnitus the intensity will gradually decrease with the passage of time.

VII. SUMMARY:

The successful management of the patient with tinnitus depends, therefore, upon the establishment of an etiologic diagnosis and in the assessment of the psychosomatic weighting factor. This should make it possible to attain the final desired goal, namely, the conversion of an uncompensated tinnitus into a psychologically compensated tinnitus. Any management of the patient with tinnitus which does not take into consideration these two aspects will usually be ineffective. Any management which is based upon a single panacea for the treatment of a symptom and not a disease will result in failure.

INTRACRANIAL LESIONS SIMULATING FRONTAL SINUS INVOLVEMENT.*

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Frontal headache is not infrequently the main symptom, and, in fact, sometimes the only symptom of an intracranial lesion. For this reason, frontal sinus disease is erroneously suspected in some of these cases, and they are referred to the rhinologist for treatment. Even though there may be a coincidental frontal sinusitis, the rhinologist should not overlook the possibility of referred pain, due to some type of intracranial lesion. While there are a great number of recognized conditions which may cause pain to be referred to the frontal area, the scope of this paper necessarily limits the discussion to some of the more tangible and demonstrable anatomic lesions.

To have a proper conception of the manner in which pain from an intracranial lesion may be referred to the frontal region, it is necessary that one have an understanding of the pain-sensitive structures of the head, the mechanism by which pain is produced and the neural pathways along which the painful impulses are transmitted. For our knowledge of these factors we are indebted to the very extensive and exhaustive work of Wolff and his various associates at the Cornell Medical Center. The review presented here is drawn largely from his many publications and also those of other investigators in this field. The material is taken from three principal sources: *1.* Moench's book on "Headache," *2.* the *Proceedings of the Association for Research in Nervous and Mental Diseases* for the year 1943, and *3.* from Wolff's monograph, "Pain."

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The pain-sensitive structures of the head are both extracranial and intracranial. The scalp—especially its arteries—is sensitive to pain. This is not true of the veins of the scalp, the cranial bone or the diploe; the periosteum of the skull is only moderately sensitive. Briefly, the intracranial pain-sensitive structures include the dural arteries, the basal parts of the cerebral arteries, parts of the dura at the base of the skull, along the olfactory groove and at the roof of the orbit, the great venous sinuses and their venous tributaries from the surface of the brain, the Vth, IXth and Xth cranial nerves and the upper three cervical nerves. The brain tissue itself, the greater part of the dura and the pia arachnoid, the lining membrane of the ventricles and the choroid plexus are insensitive to pain.

According to Wolff, a line drawn vertically over the top of the head from ear to ear divides the pain-sensitive structures into a supratentorial group and an infratentorial group. Stimulation of any of the pain-sensitive structures anterior to this line, *i.e.*, the supratentorial group, will produce pain in various regions in the anterior portion of the head, *e.g.*, the eye and the forehead. Stimulation of the pain-sensitive structures behind this line, *i.e.*, the infratentorial group, will produce pain in various regions in the posterior portion of the head, *e.g.*, the occiput and behind the ear. This is explained by the fact that the sensory division of the Vth cranial nerve supplies the superior surface of the tentorium and all the pain-sensitive structures above it, while the pain produced by stimulation of the infratentorial group is mediated by the IXth and Xth cranial nerves and the upper three cervical nerves.

Wolff has outlined six basic mechanisms by which headache may be produced from intracranial disturbances. The most important of these have to do with alterations in the cranial blood vessels, whether they be displacement of the large venous sinuses or traction on their venous tributaries, or distension and dilatation of or traction on the intracranial arteries. Less important are the inflammatory changes in and about the pain-sensitive structures of the head and direct

pressure by tumors on the cranial and cervical nerves. In certain cases, more than one of these mechanisms may be in operation and perhaps more than one pain-sensitive structure may be stimulated. Pain resulting from intracranial lesions is most always referred.

Peripheral painful stimuli initiated from the areas under consideration, on reaching the thalamus, are carried to the sensory cortex of the brain, the postcentral gyrus, where the sensation of pain is appreciated, by way of the thalamocortical radiation.

ILLUSTRATIVE CASES.

It has been stated aptly by Bailey that the first step toward establishing a diagnosis of intracranial tumor is that the possibility of its presence should occur to the physician. This statement can be enlarged to apply to other intracranial lesions.

The following cases are examples of a variety of cranial and intracranial conditions which produced pain in the frontal region.

Case 1: A white male, 55, had had recurring nasal polypi since he was of school age, which had been repeatedly removed by surgery. Eventually there was perforation of the cribriform plate. His presenting complaint, aside from nasal polypi, was severe frontal pain, especially on the right side. Later the patient had coma and minor signs in the extremities of the left side. The lesion responsible was an abscess of the right frontal lobe of the brain.

Case 2: A white female, 53, had severe pain in the right frontal region for two weeks, followed by right IIIrd nerve palsy. The lesion responsible for these symptoms was an aneurysm of the right internal carotid artery.

Case 3: A white female, 36, had had chronic recurring frontal headache since childhood. Later there was a Jacksonian convulsive component, with recent paralysis of the arm and leg. The cranial lesion was a right parasagittal cortical hemangioma.

Case 4: A white male, 56, had recently suffered from unexplained diminution of vision in the right eye and recurring pain in the right frontal region. He had a calcified aneurysm of the right internal carotid artery.

Case 5: A white male, 30, had suffered from right frontal pain extending onto the right side of his face, which had been of abrupt onset. An

involvement of the Vth nerve at the Gasserian ganglion was erroneously suspected. The actual finding was an arteriovenous aneurysm of the right parietal cortex.

Case 6: A Negro, 35, had an aneurysm of the right middle cerebral artery. The initial symptom was recurring episodes of right frontal pain, with recent rupture and intracortical hemorrhage.

Case 7: A white female, 25, had had recurring episodes of frontal pain for 10 years. Recently there had been a subarachnoid hemorrhage, with recovery, followed by partial motor aphasia and right hemiplegia. The lesion responsible was a cavernous hemangioma of the left temporal lobe.

Case 8: A white female, 47, had vague right frontal discomfort followed by swelling of the right upper lid and eventual right proptosis, resulting from an osteoma of the lateral wing of the right sphenoid bone and the lateral wall of the orbit.

Case 9: A white female, 27, had vague unexplained right frontal pain for nine years with an uncorrectable visual disturbance of the right eye. She had had a recent episode of subarachnoid hemorrhage, with recovery, but persistence of pain and visual disturbance. More recently, there had been sensory Jacksonian attacks, involving the left extremities. The brain lesion was an astrocytoma of the right frontal and temporal lobes.

Case 10: A white male, 60, had a meningioma of the left frontal lobe with skull changes of 20 years' duration. For four years, he had suffered from left frontal pain and headache. Recently there had been a convulsion with coma and aphasia.

Case 11: A white male, 67, had had a swelling on the back of his head for four years and bifrontal pain for several months. The lesion responsible was an extradural meningioma situated at the midline in the posterior parietal portion.

Case 12: A white male, 66, had a spongioblastoma multiforme of the right temporal and parietal lobes which produced bifrontal pain and visual disturbances with demonstrable homonomous hemanopsia.

Case 13: A white female, 32, had right occipital and right frontal pain with extension onto the right side of the face for 22 months, for which an hysterical origin had been suspected. Eventually, obvious cerebellar signs led to the discovery of a meningioma of the right cerebellum.

SUMMARY AND CONCLUSIONS.

1. The situation of pain-sensitive areas, intracranial and extracranial, and pathways concerned in the transmission of pain to the frontal region from intracranial lesions are reviewed and discussed.

2. Tangible, demonstrable anatomic lesions producing reference of pain to the frontal area are reported.

3. The frequency of frontal pain resulting from an intracranial lesion demands consideration of a variety of cranial

or intracranial conditions other than frontal sinus disease, as the possible causative agent.

BIBLIOGRAPHY.

BAILEY, PERCIVAL: Intracranial Tumors. Springfield, Ill.: Charles C. Thomas, 1933.

CUNNINGHAM: Textbook of Anatomy. Baltimore, Md.: William Wood and Co., 4th Ed., 1913.

MERRITT, H. H.; METTLER, F. A., and PUTNAM, T. J.: Fundamentals of Clinical Neurology. Philadelphia, Pa.: The Blakiston Co., 1947.

MOENCH, L. G.: Headache. Chicago, Ill.: The Year Book Publishers, Inc., 1947.

Proceedings, Association for Research in Nervous and Mental Diseases. Baltimore, Md.: Williams and Wilkins, 1943.

WOLFF, H. G., and WOLF, S.: Pain. Springfield, Ill.: Charles C. Thomas, 1948.

NATIONAL ASSEMBLY OF SURGEONS OF MEXICO.

Mexico City.—The meeting of the Plastic Surgery Section of the Ninth National Assembly of Surgeons will be held Nov. 19-25, 1950, at the Hospital Juarez in Mexico City.

The meeting of the American Society of Plastic and Reconstructive Surgery will be held in Mexico City, Nov. 27-29, 1950, immediately after the work of the Plastic Surgery Section of the Ninth National Assembly of Surgeons. The management of this meeting will be in charge of the American directors. Each group may execute their work according to their usual procedure.

We hope to include the meeting of the Latin American Society of Surgeons and thus make this the First International Congress of Plastic Surgery in Mexico.

For further information address the Secretary, Dr. Alfonso Duenas de la Torre.

BLOOD DYSCRASIA IN OTOLARYNGOLOGY.*

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The initial clinical symptoms of acute hematological disturbance, not infrequently, appear in the field of otolaryngology. Many patients with insidious chronic progressive blood disorder are not aware of this condition until the occurrence of protracted nasal hemorrhage, sudden vertigo and deafness or development of oral lesions which prompts them to seek the services of otolaryngologists.

The present study is based on the following cases of blood dyscrasia observed at the White Memorial Hospital, Los Angeles, from 1939 to 1949. These cases originated either *1.* in my own practice, *2.* in the outpatient department and later admitted to the hospital, or *3.* in the practice of my colleagues in other departments of the hospital in which otolaryngological consultations were sought.

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1. HEMOPHILIA.

Hemophilia is an hereditary blood disorder. The bleeders are limited to males, but females of the family transmit the disease to the male offspring. Its incidence follows the Mendelian law in that a recessive sex character is related to a single male chromosome. A complete review of the subject was given by Davidson and his associates.[1] According to the newer concept, the hemophiliac's blood lacks a clot-forming element in its plasma.[2] The blood of the hemophiliac shows normal distribution of cellular elements. His bleeding time is normal, but coagulation time is prolonged. There is no abnormality of the capillaries. Of the four hemophiliacs in this series, all were under 25 years. Each gave a history of a life-long tendency to bruise and slow, oozing type of bleeding after the slightest cut. The majority of the hemophiliacs bleed to death in childhood.

Five-year-old Kenneth had the history of epistaxis since three years of age and could not blow his nose without producing nasal bleeding. This child fell from a swing in his back yard and died from intracranial hemorrhage on the eighth day following the accident. The remaining three cases were ages 21, 23 and 25, with numerous episodes of bleeding from the mouth, lips and tongue. Each had bleeding when their teeth erupted and received medical attention. One had died in another hospital. The third could not be traced. The fourth patient, aged 23, is a semi-invalid and became a morphine habitue on account of the panhemarthrosis.

No organs are spared from hemorrhage. Hemarthrosis and hematuria are present in 90 per cent of cases.[3] Epistaxis is universal.[4] Intralingual and paralaryngeal hemorrhage is a serious complication. Three of seven received a tracheotomy.[5,6] Bleeding into the ear masquerades as acute mastoiditis. Diagnosis should not be difficult. In one out of three cases heredity may not be established.[7] Craddock and his associates[8] and Shea[9] warn that the clotting time determination may be misleading in the hemophiliac, since it takes but a small amount of thrombin in clotting when a venopuncture is made. A disastrous hemorrhage may occur when a larger

vessel is severed in the same individual. The amount of prothrombin remaining in the plasma after clotting time is a much more reliable test for hemophiliacs.

Having found the missing blood factor, this deficiency is now supplied by the antihemophilic globulin (Fraction I of Cohn) or by fresh normal plasma.[10-14] Injections of whole blood, human and animal globulin and human placental extract and thrombin preparations may also be used to tide the patient over a critical period. Much can be done if injury to hemophiliacs is avoided and proper preoperative precaution is taken. The preservation of injectable veins is an important item in the management of every hemophiliac. Nasal packing for epistaxis is dangerous. A tracheotomy set should be in easy reach when pharyngolaryngeal hematoma is suspected.

2. POLYCYTHEMIA VERA (VASQUEZ-OSLER DISEASE).

This is a slowly progressive, chronic disease of the bone marrow with many remissions and fatal termination. Its onset is insidious. Most patients date the inception of their disease back more than 10 years.[1,2] It is a disease of past middle life, some cases being familial. The highest incidence is found among the Hebrew race.[3] Under some unknown stimuli there is a striking erythroblastic activity of the bone marrow, the red cells sometimes exceeding 8,000,000 per cm. This unrestrained production of red cells is also accompanied in some cases by an increased production of immature white cells and platelets, the prognosis of which is extremely poor.[1] The proportion of cells to plasma is greatly increased in typical cases.[4] This increases the viscosity of blood which tends to elevate the blood pressure, predisposes to thrombosis and gives the blood a grossly dark color.[5]

Frequent epistaxis, headaches, postural vertigo, tinnitus, fullness in the head, mental confusion, dyspnea and cardiac embarrassment are the chief symptoms. Bleeding occurs from congestion rather than from hemorrhagic tendency. Hemorrhage and anemia occur late in the course of the disease. The physical appearance of patients suffering from polycythemia is most striking. "Red as roses in summer and blue as indigo in winter" is the description given by Osler.[6] The eyes are

bloodshot. The oral mucosa is deeply cherry. The tongue is swollen, beefsteak in color. The pharynx presents a diffuse, fiery red appearance.

There were two males and six females in this series. The youngest was 35; the oldest, 70; the majority being past 60 years of age. The following case is typical of this malady:

Mrs. E. C., aged 49. For the past year and a half before her admission to the hospital, she had numbness, substernal oppression, headaches and repeated epistaxis. During the same period she had several teeth extracted, and on one occasion bleeding of the gums continued for four days. Four months before her admission to the hospital she consulted her family physician for severe headaches and dyspnea and found that her red cell count was 7,000,000 per cm.; hemoglobin, 126 per cent. Profuse epistaxis occurred frequently. Later she was admitted to the hospital for diagnosis and remained for a week. Two venesections gave her only temporary relief.

The treatment is palliative. Radioactive phosphorus is now most popular.[7-10] X-ray has been ineffective.[11] Periodic venesections, acetyl pheny hydrazine, nitrogen mustard,[12] Fowler's solution and a diet low in iron[13-15] offer the best relief and prolong the life of the patient.

3. INFECTIOUS MONONUCLEOSIS: (INFECTIOUS LYMPHADENOSIS).

Infectious mononucleosis is a self-limited disease, probably of virus origin, occurring usually in sporadic and at times epidemic form among children and young adults (see Fig. 1). No race is spared, but Negroes are more susceptible.[1] Incubation period is seven to 15 days.

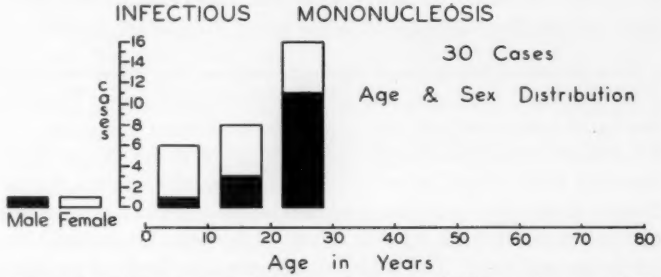

Fig. 1. Infectious mononucleosis is a disease of children and young adults. The sex is evenly divided.

Two major types are generally recognized: the pharyngeal and lymphatic varieties.[2,3] Some subdivide them as glandular, anginose and febrile, but they may merge into each other.[4-11] In the pharyngeal type the most excruciating form of sore throat is the outstanding symptom. Following the initial period of malaise, headache, fever of a few days to a week, the patient complains of acute pharyngitis. The tonsils, if present, are invariably enlarged and often coated with grayish, odorless exudate. The culture may show Vincent's organisms, various forms of streptococcus and staphylococcus. In rare instances the throat presents the clinical picture of diphtheria. Laryngeal stenosis may require a tracheotomy.[12] In the severe form there is always profound toxemia, simulating typhoid fever or an atypical pneumonia.[11,13] In the milder forms the patient may be afebrile practically throughout the entire course of the disease. The only clinical sign may be an enlarged cervical gland and a slight evidence of upper respiratory infection.

In the lymphatic type the adenopathy is the chief complaint. The cervical nodes may become tender and enlarged. At times, one must differentiate the condition from mumps. The glands of the axilla and the groin are also enlarged. Splenomegaly is present in two out of three cases. The cough may become troublesome due to enlarged mediastinal lymph nodes. Bizarre neurolgical manifestations have been recorded.[13,14] Epistaxis and hematuria are not uncommon.[15] Purpura[16,17] and jaundice may be a complication.[18-22] The disease may last a number of years.[23] So protean are the clinical manifestations of the disease it often taxes one's diagnostic acumen.

In a series of 30 cases, a correct diagnosis of mononucleosis was recorded on admission in only eight instances. Another group of seven patients was admitted without diagnosis. In the others admission diagnoses were acute pharyngitis, streptococcus sore throat, acute tonsillitis, Vincent's angina, hypertrophic adenoids, mumps, diphtheria, poliomyelitis, leucemia, pyelitis, jaundice, rheumatic fever, influenza, pneumonia and acute appendicitis. The latter diagnosis was derived probably from enlarged mesenteric lymph nodes.

The condition usually persists for several weeks with a tendency for remission and recurrence. Diagnosis is made from *1.* the clinical picture of headache, asthenia, fever of 100° to 104° F., sore throat with or without ulceration and enlarged, tender adenopathy; *2.* the presence of a positive heterophile antibody reaction (Paull-Bunnell's) in a titer 1:16 or higher, and *3.* the recognition of atypical leucocytoid lymphocytes in the circulating blood.[3,4]

During the initial stage all three of these syndromes may not be present; but if two are present in the course of the disease in the patients under 30 years of age, in a community in which upon inquiry other similar age groups have had a similar condition, the diagnosis is probably correct. The positive heterophile reaction is obtained most frequently during the second and third weeks of the disease. In a typical case there is an initial leucocytosis or normal count followed by a drop to leucopenia with a rising mononucleosis. When the diagnosis is in doubt, repeated smears and blood counts are advisable. The prognosis is good in uncomplicated cases. There are now on record nine cases of ruptured spleen resulting from this disease.[14-26] Two deaths occurred from infectious polyneuronitis, one each from nasopharyngeal hemorrhage and laryngeal edema.[13]

Diagnosis may be extremely difficult as shown in the case cited below:

A. K., a 24-year-old, white medical student, was first seen Feb. 3, 1949. His chief complaints were nasal obstruction, frontal headache, fever, 103° F. Physical signs were that of acute rhinitis, hyperplasia of the pharyngeal lymphoid tissue and cervical and submaxillary adenopathy. Because of marked prostration and high fever, he was admitted to the hospital with an admission diagnosis of influenza. The first hematological study revealed nothing of abnormal nature (see Fig. 2). The blood study and heterophile reaction on the tenth day of illness established the diagnosis. The temperature fluctuated during the first week and subsided by lysis the following week (see Fig. 3).

The treatment of infectious mononucleosis has been largely symptomatic. Recently daily intramuscular injections of serum globulin in doses of 10 cc. have been tried and found to be specific for this condition.[27]

INFECTIOUS MONONUCLEOSIS

Patient: A K						
Date:	Feb. 4	Feb. 6	Feb. 10	Feb. 11	Feb. 12	Feb. 14
Hemoglobin	96%					
Erythrocytes	4490000	4900000	4900000	5020000	4870000	4870000
Leukocytes	7900	3650	8200	10100	9900	9450
Lymphocytes	23%	37%	23%	12%	10%	12%
Monocytes	3%	7%				
Eosinophils						
Basophils						
Neutrophil Band Cells	74%	50%	31%	34%	18%	23%
Promyelocytes		2%				
Myeloblasts		2%				
Prolymphocytes			46%	53%	72%	64%

Fig. 2. Typical hemogram in infectious mononucleosis. Patient was a 24-year-old white medical student. On the fourth day of illness on Feb. 4, 1949, his cell count was normal. Feb. 6, there was leucopenia and a slight neutropenia. On the tenth day and thereafter there was prolymphocytosis until on his twentieth day of illness when the blood count became normal.

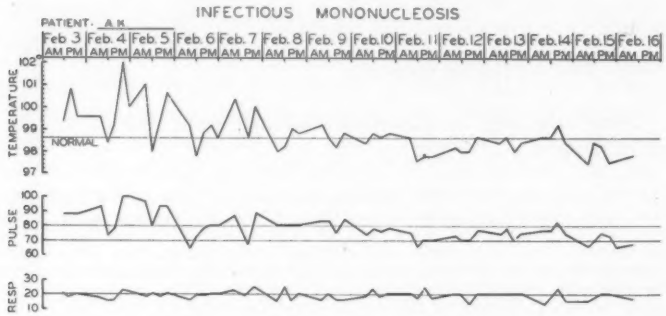

Fig. 3. The initial rise in temperature, pulse and respiration rate was followed by a gradual decline.

4. AGRANULOCYTOSIS.

Since Schultz's first paper on agranulocytosis appeared in 1922[1] interest in this disease has been unabated. During the

past 10 years the *Quarterly Cumulative Index Medicus* lists more than 740 references on this subject. The present concept of this condition may be thus summarized:[2-8]

1. The disease is an idiosyncrasy to some drug, the dosage-time factor of which has no relation to the intensity of symptoms. Women past 25 years of age are particularly susceptible. *2.* The most frequent offenders are found among the derivatives of the benzene ring, notable of which is aminopyrine. The next in order are dinitrophenal and sulfonamides. Among other drugs are thiouracil, barbiturate, various quinine derivatives, bismuth, gold, mapharsen, arsenicals, dilantone, pyribenzamine, tridione and hydantal. *3.* It also develops secondary to hyperplastic processes in pernicious anemia, leucemia, purpura, allergic reaction and acute infection. *4.* The characteristic hematologic change is brought about by suppression of maturation of granulocytes during the myeloblastic stage. The total count may not exceed 1,000 per cm. *5.* There is no alteration in red cell and platelets counts in a typical case, a factor which accounts for lack of hemorrhage. *6.* The sepsis is secondary to leucopenia and when untreated the patient succumbs to overwhelming sepsis within 36 to 72 hours of apparent onset.

The most characteristic feature of the disease is the rapidity of the course, profound toxemia and striking response to the drug withdrawal combined with antibacterial therapy in favorable cases. The disease is ushered in with alternate chills and high fever, marked prostration, malaise, headache, cervical adenopathy and necrotizing mucous membrane of the ear, nose, pharynx and oral cavity.[9,10] The soft palate, uvula and the pillars of the tonsils are extremely reddened and eroded. The process may extend to the larynx, the obstruction of which requires a tracheotomy.[11-13] The blood picture is so typical that little difficulty is to be expected in establishing a diagnosis. In the initial stage there is asymptomatic leucopenia and neutropenia. The lymphocytes consist nearly 90 per cent of the total white counts. There is no known sensitization test to determine one's idiosyncrasy, thus every trace of drug must be eliminated from the patient, and in practice

this is not always easy. At times the patient's own secretiveness defeats this very object. A young married man consulted me for the treatment of acute pharyngitis, which later was diagnosed as an agranulocytic angina. Unbeknown to me, the patient was in the habit of self-drugging off and on for a period of months with a fairly large dose of sulfonamide for a Neisserian infection which he acquired from extramarital source. On the initial visit the patient denied the drug habit. Confronted with a correct diagnosis based on hematologic study and clinical picture, the patient finally disclosed the history of the urethral infection, subsequent self-treatments and development of the recent oral lesions for which he sought my services.

The prognosis is excellent when aggressive therapy is instituted. Pentnucleotide, transfusions, pyridoxin and folic acid have enjoyed popularity; however, penicillin appears to be specific. The most important feature is its prevention,[14] and the instant withdrawal of the causative medication when diagnosed.

5. THROMBOCYTOPENIC PURPURA.

The simplest yet most comprehensive classification of purpura is given by Fowler:[1]

I. Thrombocytopenic Purpura:

A. Idiopathic or essential.

B. Secondary purpura associated with some other recognizable disease, intoxication and infections.

II. Nonthrombocytopenic Purpura:

A. Idiopathic.

1. Schönlein-Henoch or allergic purpura.

2. Purpura simplex.

B. Symptomatic purpura.

This paper is limited to the discussion of thrombocytopenic purpura, or Werlhof's disease. Pathogenesis of this condition has been thoroughly studied by competent investigators.[1-10] There were 22 cases in this series. All of the patients had in common the tendency to an abnormal bleeding into the skin and mucous membrane, a marked reduction in the number of platelets in the circulating blood, prolonged bleeding time, a defective clot retraction, a positive tourniquet test, and normal coagulation time. The disease is most frequently seen in children and youth; women suffer twice as often as men. It is rare in the Negro.[11] There is a distinct hereditary tendency. The cause of hemorrhage is not known, but is thought to be the combination of thrombocytopenia and capillary anomaly.[12,13]

Quick[14] has explained this platelet-capillary phenomena on the basis of histamine activity* and it appears to be gaining favor.[15] The course of the disease is both acute and chronic. The acute form lasts two to several weeks. It may terminate in complete restitution or in death from hemorrhage or aplastic anemia due to repeated blood loss. In the chronic form the disease shows a tendency to frequent remissions. The episodes of bleeding may last from a few days to a few weeks. The number of platelets may attain normal level during the symptomless intervals.

Epistaxis is one of the chief initial symptoms that brings the patient to a clinician. It is not unusual for the illness to appear following an acute upper respiratory infection. Without hematological investigation a diagnosis may be difficult if the cutaneous lesions are few or absent, as in the initial stage of most of the cases.[10] In 15 of the series of 22 cases, the disease was precipitated with epistaxis. A history of a tendency to bruise on slight trauma, an episode of hemorrhage from the nose, throat or vagina should arouse suspicion, or at least raise a question.

*Histamine causes capillary dilatation. One of the functions of platelets is to remove excess histamine. In so doing, platelets are rendered more susceptible to agglutination and lysis, resulting in thrombocytopenia and purpura. This concept of allergic origin explains both the thrombocytopenic and nonthrombocytopenic purpura.

At the age of 12, a Mexican boy had a tonsillectomy in another hospital. This was followed by a prolonged, oozing type of bleeding, typical of this condition. He received eight transfusions before the bleeding was controlled. He has had a tendency to bruise all his life and frequent epistaxis since two years of age, but this vital point in the clinical history was apparently overlooked in the preoperative examination.

Pregnancy adds hazards to the condition.[17] Two of my three cases spontaneously aborted and died from massive intrauterine hemorrhage; upon the third a therapeutic abortion was performed. All three had a daily epistaxis during the period of their hospitalization.

Bleeding from gums, mouth and throat are the next frequent symptoms observed. A 46-year-old male had his teeth extracted. This was followed by protracted bleeding. His only son had died following tonsillectomy and adenoidectomy in another hospital from uncontrollable bleeding, in spite of repeated transfusions.

Prognosis is generally poor in the acute form, particularly among children under 10 years of age. Death may occur in a few hours following the initial symptoms.[17,18] In the presence of shock and massive bleeding, the blood loss must be immediately replenished. X-ray and urethane have been the treatment of choice. For chronic cases, a small quantity (100 to 300 cc.) of repeated transfusions at two to three-day intervals have been recommended on the theory of stimulating the blood formation and to supply platelets. A splenectomy in chronic cases offers the best outlook. There were four patients in this series who submitted to this operative procedure. Three are living and well for seven to 10 years following the operation. One died from other causes.

6. PSEUDOHEMOPHILIA.

Pseudohemophilia (Willebrand's disease) is a rare hereditary blood dyscrasia which appears in both male and female and is transmitted by both. Unlike thrombocytopenic purpura, with which the condition at times may be confused,

there is no deficiency in the number of platelets in the circulating blood. There is no splenomegally; neither is there demonstrable defect in the clotting time nor in clot retraction. The distinct feature of the condition is a prolonged bleeding time.

Willebrand[1] originally suggested that the condition was due to anomaly of the capillaries, and his suspicion was confirmed by Macfarlane, who has shown that in pseudohemophilia there is failure of the capillary to retract upon injury.[2,7]

A number of excellent clinical reports have appeared, both in the American and German literature, under such titles as Familial Hemorrhagic Condition, Atypical Pathologic Hemorrhage, Konstitutionelle Thrombopathie, Familial Purpura, and Hereditary Pseudohemophilia.[8-11] Hewlett and Haden[12] collected 73 cases from the literature and added four cases of their own. The present series makes the total of 79 that have been recorded.

Differentiation must be made from thrombocytopenic purpura.[13,14] There is no specific treatment. Transfusion restores the blood volume, and combats a secondary anemia. Of the two patients in this report, one was a 60-year-old woman who had the history of lifelong, frequent epistaxis and ecchymosis on the slightest trauma. Her son also had a similar affection. On one occasion she had a large sublingual hematoma that made it difficult for her to talk or swallow. As she grows older, these symptoms are becoming increasingly less severe. The second was a 23-year-old woman who has had repeated spontaneous bleedings from her nose and gums. Her mother, a sister and her mother's two brothers also were similarly affected.

7. LEUCEMIA.

Leucemia is a fatal disease of irreversible, proliferative process affecting one of three types of white cells and their immature precursors in the blood-forming organs. They are lymphatic, myelogenous and monocytic leucemias. The disease appears in acute, subacute and chronic forms.[1-7] In the course

of the disease there is an increase of white cells, 50,000 leucocytes per cm. being generally accepted as a dividing line. In so-called aleucemic form, the leucocytes may not exceed 10,000 with the normal differential count. The disease is apparently on the increase.[8,9] Since 1940 the annual toll from this condition exceeds 5,000 in the United States.

The acute lymphatic leucemia occurs usually in children and youth. The acute myelogenous type occurs most frequently after 50 years of age. The acute monocytic leucemia occurs after 30 years of age. In all acute leucemias the onset is abrupt, not unlike acute infection. There is high fever, prostration, malaise, tonsillitis and headache. Cervical adenitis is a prominent feature in acute lymphatic leucemia. There is progressive anemia and its associated symptoms. Bleeding from the nose, the gums, mouth and throat is frequent and should arouse the suspicion of the clinician. Ulceromembranous stomatitis occurs in 40 to 50 per cent of cases based upon a large series of acute leucemias.[10-13] The disease runs a rapid, galloping course. In fulminating cases, the disease terminates in a few days to two or three weeks. A spontaneous remission in acute leucemia has been recorded.[14]

Of the 76 cases of leucemias in this series, 25 were classified as chronic. These chronic cases ran a milder course and occurred in older individuals. There are several features which were common to all chronic leucemias: *1.* Progressive weakness, fatiguability, pallor, dyspnea and palpitation, and secondary anemia; *2.* splenomegally, especially in chronic myelogenous forms; *3.* purpura of skin and mucous membrane; *4.* marked tendency toward remission and recrudescence; *5.* slow but steady downward course, terminating in death in two to three years in chronic myelogenous leucemia, five to six years in chronic lymphatic leucemia, and one to two and one-half years in monocytic leucemia.

The most common leucemic symptom which is of interest to the otolaryngologist is epistaxis.[15] Leucemic cutis of the nose is rare.[16] Intranasal leucemic infiltration may cause obstruction.[17,18] Protracted nasal hemorrhage may be the initial symptom that causes the suspicion of leucemia.

Intraoral lesions of leucemia appear also as hemorrhage, infiltration and ulceration.[12,13,15] The infiltration may be local or diffuse. Ulceration of the surface follows, such as an invasion from thrombosis. Hemorrhage following extraction of teeth is frequent. Sometimes the involved gums undergo ulceration with no tendency to heal and exposing necrotic bone deep in the tooth socket, covered with foul, grayish exudate, and sometimes the gums present the classical picture of Vincent's angina. Leucemic infiltration of the tonsils may simulate peritonsillitis.[19] Usually one tonsil is larger than its mate. Pain is severe, but an incision produces no pus. The infiltration may extend to the larynx, requiring a tracheotomy. A safe rule is to suspect leucemia in unilateral enlargement of tonsils until proved otherwise.

The leucemic disturbance of the ear is hemorrhage into the external and middle ears and the labyrinth, causing impairment of hearing, vertigo and tinnitus.[20] Leucemic infiltration may produce the syndrome observed in Ménière's disease.[21] If the patient survives the hemorrhagic episode, there is a new formation of the connective tissue within the labyrinth, constituting "otitis interna ossificans."[21] Paralysis of the VIIth and VIIIth cranial nerves is not rare from this source.[22]

The following otolaryngologic history was recorded on admission of 76 cases in this series:

Cervical adenopathy	29
Epistaxis	17
Dyspnea and palpitation	17
Ulcerative stomatitis	15
Bleeding gums	14
Retinal hemorrhage	8
Deafness	7
Otitis media	5
Tinnitus and vertigo	3
Hemorrhage in ear	2

The treatment of leucemia has been unsatisfactory. Those of proven palliative value are: Irradiation with X-ray,[23-25] radioactive isotope, such as radioactive phosphorus (P32),[26-30] blood transfusion,[31-35] mustard gas,[36,37] and urethane.[38,39] For acute leucemias aminopterin[40] has been found to be helpful.

8. HEREDITARY HEMORRHAGIC TELANGIECTASIA. [RENDU-OSLER-WEBER DISEASE.]

This is an hereditary blood disorder involving both sexes, the lesions appearing in the mucous membrane and the skin as ruby-red telangiectasia. There is no disturbance either in plasma, cells or in their chemical behavior. The primary site of the lesion is in the anomalies of the vascular walls.[1-10] Stock[11] estimated, in 1944, that from a number of reported cases there were probably 175 affected families involving 1,000 members. Mortality ranges from 4 to 6 per cent.[8,12]

The M. family (see Fig. 4): The bleeder in the first generation and another in the second are dead from unknown causes not associated with

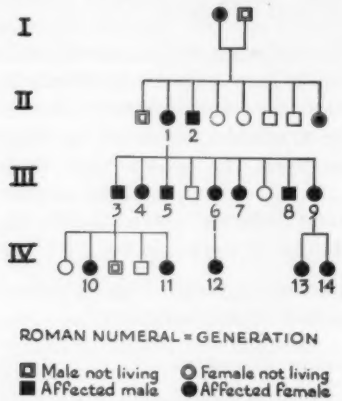

Fig. 4. The bleeder in the first and another in the second generation are dead from causes not associated with the blood disorder. The oldest is a 75-year-old widow. Her sons, aged 52, 50 and 38, and daughters, aged 51, 45, 42 and 40, show well marked telangiectatic lesions. Among the offspring in the fourth generation there were five girls, ages 22-13, with bleeding tendencies.

hemorrhagic disorders. Nine were personally examined by me. Three are in other states, but furnished detailed personal history. The oldest is a 75-year-old widow. Her sons, aged 52 and 50, and daughters, aged 51, 45 and 40, show well marked telangiectatic lesions on their lips, tongue, pharynx, cheeks, nasal septum, turbinates and fingertips. Among the offspring in the fourth generation there were two married women, each aged 20, and three girls, aged 22, 17 and 13. These showed incipient pin-sized cutaneous lesions to fairly well formed ruby-red, spider-shaped telangiectasis of the mucous membrane of the nose and oral cavity.

Epistaxis is the initial symptom. Bleeding begins without any appreciable cause or following nose blowing, sneezing or coughing. Three had developed nasal bleeding before their tenth year. In others, it came on after the thirtieth year. Emotional disturbances play a distinct part in precipitation of epistaxis. Joking, laughing and crying are often predisposing factors. Bleeding may occur in any part of the body. Blood loss may be so insignificant that the patient seeks no medical care or it may be so profuse, occurring at frequent intervals, that well-marked signs of secondary anemia are present. Diagnosis presents no difficulty. There is no known specific remedy. Aside from control of hemorrhage and secondary anemia, oral administration of hesperidian methyl chalcone appears to be helpful.[13]

COMMENTS.

Hemorrhagic disease of the newborn is a pediatric problem and not included in this study. Patients presenting the Plummer-Vinson syndrome (evidences of microcytic, hypochromic anemia, achlorhydria, nutritional disorder, glossitis, cheilitis and splenomegaly) were occasionally seen in the dispensary, but they are not included in this paper. Probably subclinical cases of avitaminosis were overlooked, but patients suffering from scurvy with bleeding from the gums were not encountered.

SUMMARY.

1. One hundred fifty-nine cases of various blood dyscrasia in otolaryngologic practice are reviewed.

2. One cannot place too much importance on a well elicited clinical history, including the tendency for bleeding in the family and patient, a careful search for evidence of nutritional deficiency and any deviation from normal in the hematologic investigation.

3. Repeated blood examinations are sometimes necessary before a diagnosis is made. It is also an indispensable procedure in determination of the therapeutic efficacy and final prognosis.

REFERENCES ON HEMOPHILIA.

1. Davidson, C. S.; Epstein, R. D.; Miller, G. F., and Taylor, F. H. L.: Hemophilia: A Clinical Study of 40 Patients. *Blood*, 4:97-119, Feb., 1949.

2. Quick, A. J.: Modern Concept of Coagulation of Blood. *Marquette Med. Rev.*, 13:89-94, Apr. 19, 1948.

Quick, A. J.: Pediatric Aspects of Hemophilia. *Pediatrics*, 3:312-317, Mar., 1949.

Quick, A. J.: Quantitative Studies on the Coagulation Defect in Hemophilia. *Fed. Proc.*, 6:284, 1947.

3. Taylor, F. H. L.: Hemophilia. *Amer. Prac.*, 2:811-813, Aug., 1948.

4. Birch, C. L.: Hemophilia. Univ. of Ill. Press, 1937.

5. Macfarlane, R. G.: Discussion on Throat and Nose Manifestations of Blood Diseases by W. M. Mollison. *Proc. Roy. Soc. Med.*, 35:401-402, Apr., 1942.

6. Baird, K. H., and Fox, M. S.: Severe Sublingual and Paratracheal Hemorrhage in Hemophilia with Recovery Following Tracheotomy. *Jour. Pediat.*, 23:90-94, July, 1943.

7. Wintrobe, M. M.: Clinical Hematology. Philadelphia: Lea & Febiger, 2nd Ed., 1946.

8. Craddock, C. G., Jr.; Fenninger, L. D., and Simmons, Bradford: Hemophilia. *Ann. Surg.*, 128:888-903, Nov., 1948.

9. Shea, J. J., and Shea, J. J., Jr.: Hematology. The Laryngoscope, 59:693-708, July, 1949.

10. Howell, W. H.: Hemophilia. *Bull. N. Y. Acad. Med.*, 15:3-26, Jan., 1939.

11. Taylor, F. H. L.; Davidson, C. S.; Tagnon, H. J.; Adams, M. A.; MacDonald, A. H., and Minot, G. R.: Studies in Blood Coagulation: The Coagulation Properties of Certain Globulin Fractions of Normal Human Plasma *in Vitro*. *Jour. Clin. Invest.*, 24:698-703, Sept., 1945.

12. Minot, G. R.; Davidson, C. S.; Lewis, J. H.; Tagnon, H. J., and Taylor, F. H. L.: The Coagulation Defect in Hemophilia: The Effect, in Hemophilia, of Parenteral Administration of a Fraction of the Plasma Globulins Rich in Fibrinogen. *Jour. Clin. Invest.*, 24:704-707, Sept., 1945.

13. Minot, G. R., and Taylor, F. H. L.: Hemophilia: The Clinical Use of Antihemophilic Globulin. *Ann. Int. Med.*, 26:363-367, Mar., 1947.

14. Alexander, B., and Landwehr, G.: Studies of Hemophilia. The Control of Hemophilia by Repeated Infusion of Normal Human Plasma. *Jour. A. M. A.*, 138:174-179, Sept. 18, 1948.

REFERENCES ON POLYCYTHEMIA VERA.

1. Tinney, W. S.; Hall, B. E., and Giffin, H. Z.: The Prognosis of Polycythemia Vera. *Proc. Staff Meet. Mayo Clin.*, 20:306-310, Aug. 22, 1945.

2. Hodgson, J. R.; Good, C. A., and Hall, B. E.: Roentgenographic Aspects of Polycythemia Vera. *Proc. Staff Meet. Mayo Clin.*, 21:152-157, Apr. 3, 1946.

3. Dameshek, W., and Hanstell, H. H.: Diagnosis of Polycythemia Vera. *Ann. Int. Med.*, 13:1360-1387, Feb., 1940.

4. Hayden, Russell L.: Principle of Hematology. Philadelphia: Lea & Febiger, 3rd Ed., 1946.

5. Stanton, J., and Scharf, L. E.: Polycythemia Vera. *Med. Bull. Vet. Adm.*, 20:453-454, Apr., 1944.

6. Osler, W.: Chronic Cyanosis with Polycythemia and Enlarged Spleen; New Clinical Entity. *Med. Class.*, 4:254-275, Nov., 1939.

7. Hall, B. E.; Watkins, C. H.; Hargraves, M. M., and Giffin, H. Z.: Radioactive Phosphorus in the Treatment of Polycythemia Vera. *Amer. Jour. Med. Sci.*, 209:712-717, June, 1945.

8. Reinhard, E. H.; Moore, C. V.; Bierbaum, O. S., and Moore, S.: Radioactive Phosphorus P^{32} as a Therapeutic Agent. *Jour. Lab. and Clin. Med.*, 31:107-215, Feb., 1946.

9. Laurence, J. H.: Control Polycythemia. *Jour. A. M. A.*, 141:13-18, Sept. 3, 1949.

10. Towle, R. A.; Coulson, F. H., and Fowler, W. M.: Polycythemia Vera. *Jour. Iowa Med. Soc.*, 35:79-82, Mar., 1945.

11. Dameshek, W.: Editorial, Treatment of Polycythemia. *Blood*, 1:256, May, 1946.

12. Wilkinson, J. F., and Fletcher, F.: Effect of B-Chlorethylamine Hydrochlorides (Mustard Gas). *Lancet*, 2:540-545, Oct. 11, 1947.

13. Jacobson, L. O.; Spurr, C. L.; Barron, E. S. G.; Smith, T.; Lushbaugh, C., and Dick, G. F.: Nitrogen Mustard Therapy. *Jour. A. M. A.*, 132:263-271, Oct. 5, 1946.

14. Richardson, W., and Robbins, L. L.: The Treatment of Polycythemia Vera by Spray Irradiation. *N. Eng. Jour. Med.*, 238:78-82, Jan. 15, 1948.

15. Fishbein, Morris: "Queries and Minor Notes": Polycythemia Vera. *Jour. A. M. A.*, 141:1109, Dec. 10, 1949.

REFERENCES ON INFECTIOUS MONONUCLEOSIS.

1. Blain, A., III, and Heide, E. C. Vonder: Infectious Mononucleosis in the Negro. *Amer. Jour., Med. Sci.*, 209:587-595, May, 1945.

2. Ray, E. S., and Cecil, R. C.: Infectious Mononucleosis in Negro: Report of Three Cases, with One Complicated by Sickle Cell Anemia. *South. Med. Jour.*, 37:543-546, Oct., 1944.

3. Bethell, F. H.; Sturgis, C. C.; Rundles, R. W., and Myers, M. C.: Progress in Internal Medicine. Blood: A Review of Recent Literature. *Arch. Int., Med.*, 77:80-119, Jan., 1946.

4. Sturgis, C. C.: Some Diseases Associated with Changes in White Blood Cells. *Hawaii Med. Jour.*, 7:133-137, Nov.-Dec., 1947.

Sturgis, C. C.: Leucemia, Infectious Mononucleosis and Agranulocytosis. *Bull. Vanc. Med. Assn.*, 23:277-284, July, 1947.

5. Gendel, B. R., and Cottrell, J. E.: Infectious Mononucleosis. Review. *Amer. Prac.*, 2:472-478, Mar., 1948.

6. Gorham, L. Whitington: Infectious Mononucleosis in Cecil's Practice of Medicine (in R. L. Cecil's Textbook of Medicine, 7th Ed., Philadelphia: Saunders, 1947).

7. Bernstein, A.: Infectious Mononucleosis. *Medicine*, 19:85-159, Feb., 1940.

8. Christian, Henry A.: Principle and Practice of Medicine. New York: Appleton-Century, 16th Ed., 1947.

9. Tidy, Sir Henry: Infectious Mononucleosis. *Blood*, 3:823-829, Aug., 1948.

10. Mitchell, R. H., and Zetzel, L.: Infectious Mononucleosis in Army. *War Med.*, 5:356-360, June, 1944.

11. Contratto, A. W.: Infectious Mononucleosis: Study of 196 Cases. *Arch. Int. Med.*, 73:449-459, June, 1944.

12. Lemierre, A.; Morin, M., and Alison, M.: Serious Complication of Asphyxia in Two Cases of Infectious Mononucleosis. *Bull. et Mem. Soc. Med. d. hop. de Paris*, 64:51-54, Jan. 9-16, 1948.

13. Custer, R. P., and Smith, E. B.: The Pathology of Infectious Mononucleosis. *Blood*, 3:830-857, Aug., 1948.

14. Ricker, W.; Blumberg, A.; Peters, C. H., and Widerman, A.: The Association of the Guillian-Barre Syndrome with Infectious Mononucleosis. *Blood*, 2:217-226, May, 1947.

15. Damashek, W.: Editorials. *Blood*, 1:357, 1946.

16. Dameshek, W., and Grassi, M. A.: Infectious Lymphadenosis (Mononucleosis) and Thrombocytopenic Purpura; Recovery After Splenectomy. *Blood*, 1:339-342, 1946.

17. Lloyd, P. C.: Acute Thrombocytopenic Purpura in Infectious Mononucleosis; Report of Case. *Amer. Jour. Med. Sci.*, 207:620-626, May, 1944.

18. Bethell, F. H.; Sturgis, C. C.; Rundles, R. W., and Meyers, M. C.: Infectious Mononucleosis. Blood. *Arch. Int. Med.*, 77:80-119, Jan., 1946.

19. Spring, M.: Jaundice in Infectious Mononucleosis. *Bull. U. S. Army Med. Dept.*, 81:102-113, Oct., 1944.

20. Boger, W. P.: Infectious Mononucleosis of Unusual Severity, with Review of Jaundice Cases Occurring in This Disease. *South. Med. Jour.*, 37:546-551, Oct., 1944.

21. Monat, H. A.: Infectious Mononucleosis Simulating Acute Infectious Jaundice. *Rev. Gastroenterol.*, 11:114-115, Mar.-Apr., 1944.

22. Morris, M. H.; Robbins, A., and Richter, E.: Acute Infectious Mononucleosis with Hepatitis; Presentation of Two Cases. *N. Y. State Jour. Med.*, 44:1579-1581, July 15, 1944.

23. Isaacs, R.: Chronic Infectious Mononucleosis. *Blood*, 3:858-861, Aug., 1948.

24. Brien, F. S.: Infectious Mononucleosis — Complications. *Canad. Med. Assn. Jour.*, 56:499-502, May, 1947.

25. Ziegler, E. E.: Infectious Mononucleosis; Report of Fatal Case with Autopsy. *Arch. Pathol.*, 37:196-201, Mar., 1944.

26. Vaughn, S. L.; Regan, J. S., and Terplan, K.: Infectious Mononucleosis Complicated by Spontaneous Rupture of the Spleen and Central Nervous System Involvement. *Blood*, 1:334-338, July, 1946.

27. Bower, A. G.: In personal communication.

REFERENCES ON AGRANULOCYTOSIS.

1. Schultz, W.: Uber eigenartige Halserkrank ungen. *Deutch. med. Wchnschr.*, 48:1495, 1922.

2. Sturgis, C. C.: Hematology. Springfield, Ill.: C. C. Thomas Co., 1949.

Sturgis, C. C.: Some Diseases Associated with Changes in White Blood Cells. *Hawaii Med. Jour.*, 7:133-137, Nov.-Dec., 1947.

Sturgis, C. C.: Discussion of Some Aspects of Leucemia, Infectious Mononucleosis and Agranulocytosis. *Bull. Vanc. Med. Assn.*, 23:277-284, July, 1947.

Sturgis, C. C.: Certain Diseases Associated with Changes in the Circulating Leucocytes. *Jour. Iowa State Med. Soc.*, 29:1-6, Jan., 1939.

3. Bethell, F. H.; Sturgis, C. C.; Rundles, R. W., and Meyers, M. C.: Blood: Review of Recent Literature. *Arch. Int. Med.*, 76:239-254, Oct., 1945.

4. Bethell, F. H.; Sturgiss, C. C.; Mallery, O. T., Jr., and Rundles, R. W.: Blood: Review of Recent Literature. *Arch. Int. Med.*, 74:131-152, Aug., 1944.

5. Dameshek, W.: Agranulocytosis (in Oxford Med., Vol. 2, Part 3, New York: Oxford Univ. Press, 1944).

6. Wintrobe, W. W.: Clinical Hematology. Philadelphia: Lea & Febiger, 1946.

7. Fowler, W. M.: Hematology. New York: Paul B. Hoeber, 2nd Ed., 1949.

8. Haden, R. L.: Principles of Hematology. Philadelphia: Lea & Febiger, 1940.

9. Oppikofer, E. K.: Necrotic Otitis and Rhinitis in Granulocytosis. *Ztschr. f. Hals., Nasen. u. Ohrenh.*, 48:495-502, 1942.

10. Heindl, A.: With Peculiar Changes in Nasal Mucosa. *Monatsschr.-Ohrenh.*, 74:630-637, Dec., 1940.

11. Pothoven, W. J.: Bilateral Laryngeal Necrosis. *Arch. f. Ohrenh., Nasen. u. Kehlkopfh.*, 147:196-201, 1940.

12. Smith, M. T.: Involvement of Larynx in Agranulocytosis Angina: Report of Three Cases. The Laryngoscope, 44:320-326, Apr., 1934.

13. Story, G. B. T., and McCabe, A. F.: Case of Infectious Mononucleosis Requiring Tracheotomy. *Brit. Med. Jour.*, 2:652, Nov. 2, 1946.

14. Bruck, E.: Agranulocytosis in Childhood. *Amer. Jour. Dis. Child.*, 73:186-194, Feb., 1947.

REFERENCES ON LEUCEMIA.

1. Sturgis, C. C.: Hemorrhagic Disorders. *Hawaii Med. Jour.*, 7:41-45, Sept.-Oct., 1947.

Sturgis, C. C.: Leucemia, Infectious Mononucleosis and Agranulocytosis. *Bull. Vanc. Med. Assn.*, 23:277-284, July, 1947.

Sturgis, C. C.: (In R. L. Cecil: Textbook of Medicine, 7th Ed., Philadelphia: Saunders, 1947.)

Sturgis, C. C.: Hematology. Springfield, Ill.: Charles C. Thomas, 1949.

2. Christian, H. A.: The Principle and Practice of Medicine. New York: Appleton-Century, 16th Ed., 1947.

3. Wintrobe, M. M.: Clinical Hematology. Philadelphia: Lea & Febiger, 2nd Ed., 1946.

4. Fowler, W. M.: Hematology. New York: P. B. Hoeber, 2nd Ed., 1949.

5. Haden, R L.: Hematological and Clinical Characteristics of Leucemia. *Amer. Jour. Radiol.*, 55:387-392, Apr., 1946.

6. Torkner, C. E.: Leucemia and Allied Disorders. New York: The MacMillan Co., 1938.

7. Kirshbaum, J. D., and Preuss, F. S.: Leucemia: Clinical and Pathological Study of 123 Fatal Cases in a Series of 14,400 Necropsies. *Arch. Int. Med.*, 71:777-792, June, 1943.

8. Dameshek, W.: Editorial: Chemotherapy of Leucemia and Related Diseases. *Blood*, 1:446-447, 1946.

DAMESHEK, W.: Editorial: Is Leucemia Increasing? *Blood*, 2:101, 1947.

9. SACKS, M. S., and SEEMAN, I.: A Statistical Study of Mortality from Leucemia. *Blood*, 2:1-14, 1947.

10. BURKETT, L. W.: Histopathology Explanation for the Oral Lesions in the Acute Leucemias. *Amer. Jour. Orthodontia*, 30:516-523, Sept., 1944.

11. ASELTINE, L. F.: Monocytic Leucemia with Oral Manifestations; Report of Case. *Jour. Oral Surg.*, 2:266-267, July, 1944.

12. BOYD-COOPER, B.: Ulcerative Stomatitis in a Case of Subacute Monocytic Leucemia. *Brit. Dent. Jour.*, 76:1-4, Jan. 7, 1944.

13. BARNFIELD, W. F.: Leucemia and Dental Procedures: A Review of the Literature and Study of 123 Cases. *Amer. Jour. Orthodontics* (Oral Surg. Sec.), 31:329-353, June, 1945.

14. BIRGE, R. F.; JENKS, A. L., JR., and DAVIS, S. K.: Spontaneous Remission in Acute Leucemia. *Jour. A. M. A.*, 140:589-592, June 18, 1949.

15. LOVE, A. A: Manifestations of Leucemia Encountered in Otolaryngologic and Stomatologic Practice. *Arch. Otolaryngol.*, 23:173-221, Feb., 1936.

16. WIENER, K.: Skin Manifestations of Internal Disorders. St. Louis: C. V. Mosby Co., 1947.

17. HAFFLY, G. N., and SCHIPFER, L. A.: Subacute Monocytic Leucemia with Nasal Manifestations. *Arch. Otolaryngol.*, 31:858-862, May, 1940.

18. HATCH, F. N.: Leucemia as a Cause of Nasal Obstruction. *Calif. and West. Med.*, 41:23-26, July, 1944.

19. BALLENGER, H. C.: Diseases of Nose, Throat and Ear. Philadelphia: Lea & Febiger, 9th Ed., 1947.

20. KERRISON, P. D.: Diseases of the Ear. New York: J. B. Lippincott, 3rd Ed., 1923.

21. FRASER, J. S.: Affections of Labyrinth and VIIIth Nerve in Leucemia. *Ann. Otol., Rhinol. and Laryngol.*, 37:361-371, Mar., 1928.

22. BRUNNER, H.: Changes of Temporal Bone in Leucemia and Osteitis Fibrosa. *Arch. Otolaryngol.*, 39:1-13, Jan., 1944.

23. ALT, H. L.: Treatment of Leucemia. *Med. Clin. N. Amer.*, 28:187-200, Jan., 1944.

24. POPP, W. C.: Roentgen Therapy for Myelogenous Leucemia. *Proc. Staff Meet. Mayo Clin.*, 21:331-335, Aug., 1946.

POPP, W. C., and WATKINS, C. H.: Roentgen Therapy for Leucemias. *Med. Clin. N. Amer.*, 30:799-810, July, 1946.

25. FRIEDMANN, A. B., and MEYER, L. M.: Observations on Over 100 Cases of Myelogenous and Lymphatic Leucemia. *Radiol.*, 44:341-343, Apr., 1945.

26. WEBSTER, C.: Use of Radioactive Phosphorus in the Treatment of Leucemia. *Tex. Rep. Biol. and Med.*, 2:83-88, 1944.

27. WARREN, S., and COWING, R. F.: The Retention of Radioactive Phosphorus When Administered in Different Chemical Forms. *Cancer Research*, 4:113-115, Feb., 1944.

28. HEMPELMANN, L. A., JR., and Others: Hematologic Complications of Therapy with Radioactive Phosphorus. *Jour. A. M. A.*, 124:735, Mar. 11, 1944.

29. REINHARD, E. H.; MOORE, C. V.; BIERBAUM, O. S., and MOORE, S.: Radioactive Phosphorus as Therapeutic Agent. *Jour. Lab. and Clin. Med.*, 31:107-215, Feb., 1946.

30. LAWRENCE, J. H.: Use of Isotopes in Medical Research. *Jour. A. M. A.*, 134:219-225, May 17, 1947.

31. HENSTELL, H. H.; HENSTELL, I. S.; SALISBURY, P. F., and MILLER, J. H.: Replacement Transfusions in the Treatment of Acute Leucemia. *Ann. West. Med. and Surg.*, 3:371-379, Nov., 1949.

32. BESSIS, M.: Use of Replacement Transfusions in Diseases Other Than Hemolytic Disease of Newborn. *Blood*, 4:324, Apr., 1949.

BESSIS, M., and BERNARD, J.: Concerning Treatment of Acute Leucemia by Exchange Transfusion. *Bull. et mem. Soc. Med. d. Hop. de* Paris, 29:14, 1948.

33. SCHWIND, J. L.: Partial Maturation of Leucemic Myeloblasts Following Fresh Plasma Transfusions. *Amer. Jour. Med. Soc.*, 213:170-175, Feb., 1947.

34. BESSIS, M.: Blood Substitution Applied to Medical Research. *Rev. d'Hemat.*, 3:118, 1948.

35. THIERSCH, J. B.: The Failure of Penicillin and Direct Blood Transfusion in the Treatment of Acute Leucemia. *Med. Jour. Australia*, 2:743-744, Nov. 23, 1946.

36. GOODMAN, L. S., *et al.*: Nitrogen Mustard Therapy for Hodgkin's Lymph Sarcoma, Leucemia. *Jour. A. M. A.*, 132:126-132, Sept. 21, 1946.

37. RHOADS, C. P.: Nitrogen Mustards in the Treatment of Neoplastic Disease. *Jour. A. M. A.*, 131:656-658, June 22, 1946.

38. OSGOOD, E. E., and CHU, I. T.: The Effect of Urathene on the Nuclear Morphology of Cells of the Granulocyte Series as Observed in Marrow Cultures and Leucemic Blood. *Blood*, 3:911-917, Aug., 1948.

39. CRESKOFF, A. J.; FITZ-HUGH, T., JR., and FROST, J. W.: Urethane Therapy in Leucemia. *Blood*, 3:896-910, Aug., 1948.

40. HUNTER, O. B., JR., and CARROLL, G. J.: Aminopterin (4-Amino-Pterol-Glutamic Acid) Therapy in Leucemias. *Ann. West. Med. and Surg.*, 3:380-384, Nov., 1949.

REFERENCES ON PSEUDOHEMOPHILIA.

1. VON WILLEBRAND, E. A.: Hereditary Pseudohemophilia; Description; Previously Observed Cases. *Finska lak.-sollsk. handl.*, 68:87-112, Feb., 1926.

VON WILLEBRAND, E. A.: Uber hereditare Pseudohamophilie. *Acta med. Scandinav.*, 76:521-550, 1931.

2. ESTREN, S.; SANCHEZ MEDAL, L., and DAMESHEK, W.: Pseudohemophilia. *Blood*, 1:504-533, Nov., 1946.

3. QUICK, A. J.: The Hemorrhagic Diseases and the Physiology of Hemostasis. Springfield, Ill.: Charles C. Thomas, 1942.

4. MINOT, G. R.: Familial Hemorrhagic Condition Associated with Prolongation of Bleeding Time. *Amer. Jour. Med. Sci.*, 175:301-306, Mar., 1928.

5. MACFARLANE, R. G.: Critical Review: Mechanism of Hemostasis. *Quar. Jour. Med.*, 10:1-29, Jan., 1941.

6. CLOUGH, P. W.: Editorial: Pseudohemophilia. *Ann. Int. Med.*, 26: 459-464, 1947.

7. SHIVELY, R. L.: Battle Wound of Thigh Complicated by Gas Gangrene in Case of Hereditary Hemorrhagic Thrombasthenia. *Ohio State Med. Jour.*, 42:151-153, Feb., 1946.

8. BUCKMAN, T. E.: Atypical Pathologic Hemorrhage in Early Life. *Amer. Jour. Med. Sci.*, 175:307-312, Mar., 1928.

9. VON WILLEBRAND, E. A., and JURGENS, R.: Uber ein neues vererbbares Blutungsübel: Die konstitutionelle thrombopathie. *Deutch. Arch. f. klin. Med.*, 175:453-483, 1933.

10. BAILEY, F. R., and MCALPIN, K. R.: Familial Purpura; Report of Two Cases. *Amer. Jour. Med. Sci.*, 190:263-268, Aug., 1945.

11. FOWLER, W. M.: Hereditary Pseudohemophilia *Amer. Jour. Med. Sci.*, 193:191-198, Feb., 1937.

12. HEWLETT, J. S., and HADEN, R. L.: Hereditary Pseudohemophilia. *Cleveland Clin. Quar.*, 15:118-124, 1948.

13. STURGIS, C. C.: Hematology. Springfield, Ill.: C. C. Thomas, 1949.

14. FOWLER, W. M.: Hematology. Philadelphia: Lea & Febiger, 2nd Ed., 1949.

REFERENCES ON TELANGIECTASIA.

1. RENDU, M.: Epistaxis repetees chez unsujet porteur de petits angiomas cutanes et muquez. *Bull. et. Mem. Soc. Med. d. Hop. de Paris*, 13: 731, Oct. 23, 1896.

2. OSLER, W.: On Multiple Hemorrhagic Telangiectases with Recurring Hemorrhages. *Quar. Jour. Med.*, 1:53, Oct., 1901.

OSLER, W.: On a Family Form of Recurring Epistaxis, Associated with Multiple Telangiectases of the Skin and Mucous Membranes. *Bull. Johns Hopkins Hosp.*, 12:333, Nov., 1901; *Med. Class.*, 4:243-253, Nov., 1939.

OSLER, W.: On Multiple Hereditary Telangiectases with Recurring Hemorrhages. *Med. Class.*, 4:276:283, Nov., 1939.

OSLER, W.: On Telangiectasis Circumscripta Universalis. *Bull. Johns Hopkins Hosp.*, 18:401, Oct., 1907.

3. WEBER, F. P.: A Case of Multiple Hereditary Developmental Angiomata (Telangiectases) of the Skin and Mucous Membranes Associated with Recurring Hemorrhages. *Lancet*, 1:43, Feb. 7, 1908.

WEBER, F. P.: Multiple Hereditary Developmental Angiomata (Telangiectases) of the Skin and Mucous Membranes with Recurring Epistaxis. *Proc. Roy. Soc. Med.* (Clin. Sec.), 1:65, Jan. 10, 1908.

WEBER, F. P.: Multiple Hereditary Developmental Angiomata (Telangiectases) of the Skin and Mucous Membranes Associated with Recurring Hemorrhages. *Lancet*, 2:160, July, 1907.

WEBER, F. P.: Hereditary Hemorrhagic Telangiectasia. *Proc. Roy. Soc. Med.* (Clin. Sec.), 17:2, 1907-1908.

WEBER, F. P.: Developmental Telangiectatic Hemorrhage and So-Called "Telangiectasia"—Familial and nonfamilial. *Brit. Jour. Child. Dis.*, 21:198, July-Sept., 1924.

WEBER, F. P.: Hemorrhagic Telangiectasia of the Osler Type—"Telangiectatic Dysplasia." An Isolated Case, with Discussion on Multiple Pulsating Stellate Telangiectases and Other Striking Hemangiectatic Conditions. *Brit. Jour. Dermat.*, 48:182, Apr., 1936.

WEBER, F. P.: Telangiectasia of the Rendu-Osler Type with Camptodactylia and Muscular Atrophy in the Hands. *Proc. Roy. Soc. Med.*, 31: 258, Nov. 18, 1937.

4. HANES, F. M.: Multiple Hereditary Telangiectases Causing Hemorrhage (Hereditary Hemorrhagic Telangiectasia). *Bull. Johns Hopkins Hosp.*, 20:63-72, 1909.

5. STEINER, W. R.: Hereditary Hemorrhagic Telangiectasia: With Report of Three Families and a Review of Those Previously Recorded. *Arch. Int. Med.*, 19:194-219, Feb. 17, 1917.

6. Goldstein, H. I.: Hereditary Hemorrhagic Telangiectasia with Recurring (Familial) Hereditary Epistaxis with a Report of 11 Cases in One Family. *Arch. Int. Med.*, 27:102, Jan., 1921.

Goldstein, H. I.: Familial Epistaxis. *Amer. Physician*, 27:426, June, 1922.

Goldstein, H. I.: Familial Epistaxis with and without Skin Lesions. *Jour. Med. Soc. N. Jer.*, 19:50-51, Feb., 1922.

Goldstein, H. I.: Hereditary Epistaxis with and without Hereditary (Familial) Multiple Hemorrhage Telangiectasia. *Jour. Med. Soc. N. Jer.*, 28:309-327, Apr., 1931.

Goldstein, H. I.: Hemorrhages in Rendu-Osler-Weber's Disease. *Med. Rev. of Rev.*, 37:213, 1931.

Goldstein, H. I.: Goldstein's Heredofamilial Angiomatosis (Telangiectasia) with Recurring Familial Hemorrhages (Rendu-Osler-Weber's Disease) Case Reports. *Del. State Med. Jour.*, 3:161-172, Sept., 1931.

Goldstein, H. I.: Hereditary Multiple Telangiectasia, Goldstein's Heredofamilial Angiomatosis with Familial Hemorrhages or Rendu-Osler-Weber's Disease. *Arch. Dermat. and Syph.*, 26:282-308, Aug., 1932.

Goldstein, H. I.: Heredofamilial Angiomatosis Telangiectasia with Recurring Hemorrhages. *Tran. Amer. Ther. Soc.*, 32:47-64, 1932.

Goldstein, H. I.: Epistaxis without Telangiectasis. *Lancet*, 1:116, Jan. 14, 1933.

Goldstein, H. I.: Heredofamilial Angiomatosis (Telangiectasia with Recurring Hemorrhages — Nonhemophilic, Nonpurpuric and Nonthrombopathic). *Internat. Clin.*, 2:43-56, June, 1934.

Goldstein, H. I.: Goldstein's Heredofamilial Bleeding (Nonpurpuric and Nonhemophilic). *Jour. A. M. A.*, 100:1453, May 6, 1933.

Goldstein, H. I.: Unusual Hematemesis or Gastrorrhagia Hereditary Hemorrhagic Telangiectatic Dysplasia with Gastrostaxis or Stomachorrhagia (Goldstein's Hematemesis). *Amer. Jour. Digest. Dis. and Nutrition*, 5:115-116, Apr., 1938.

7. Larrabee, R. C., and Littman, D.: Hereditary Hemorrhagic Telangiectasia. *N. Eng. Jour. Med.*, 207:1177-1182, Dec. 29, 1932.

8. Houser, K. M.: Hereditary Hemorrhagic Telangiectasia. *Ann. Otol., Rhinol. and Laryngol.*, 43:731-738, Sept., 1934.

9. Sterman, M. M., and Scal, J. C.: Familial Telangiectasia with Recurring Epistaxis. *N. Y. State Jour. Med.*, 37:287-292, Feb. 1, 1937.

10. Barrock, J. J.: Hereditary Hemorrhagic Telangiectasia. *Wis. Med. Jour.*, 43:805-814, Aug., 1944.

11. Stock, M. F.: Hereditary Hemorrhagic Telangiectasia, Review of Literature and Report of Cases. *Arch. Otol.*, 40:108-114, Aug., 1944.

12. Voyles, G. Q., and Ritchey, J. O.: Hereditary Hemorrhagic Telangiectasia. *Ann. Int. Med.*, 22:730-736, May, 1945.

13. Eli Lilly Company, Product of.

THE LOCAL APPLICATION OF "SULFAMYLON" [PARA-(AMINOETHYL)-BENZENE SULFONAMIDE HYDROCHLORIDE] IN OTITIS EXTERNA AND CHRONIC OTITIS MEDIA.*

A Second Report of 164 Infections in 122 Patients.†

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Otolaryngologists who practice in the South and Southwest of the United States have come to accept otitis externa as one of their perennial problems. I would not imply that it is a problem which has been solved, but certainly we have come a long way toward its solution. Within the last decade the literature of otitic infections in general and of otitis externa in particular has begun to approach adequacy: comprehensive studies in classification have been made; fungi have been assigned their proper rôle as etiologic factors; the rôle of other micro-organisms has come to be appreciated, and finally, methods of treatment are becoming a good deal more rational. When one examines one's old records, as I recently examined mine, it becomes very clear indeed how much progress has been made.

It has been a source of considerable surprise to me to learn from certain comments on my preliminary publication on the subject of sulfamylon [para-(aminoethyl)-benzene sulfonamide hydrochloride] in the treatment of otitis externa and chronic otitis media[1] that I am credited with recommending this agent in the management of all otitic infections. It is difficult to know how such an impression could arise from a

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paper which bears a title specifically limiting the use of the drug to the two conditions mentioned. Sulfamylon is no panacea and was not proposed as such. It is doubtful that a single agent will ever be found which will cure all varieties of otitic infection. Sulfamylon should certainly not be used for any such purpose. It would not be indicated in the fungoid types of infection, for instance; though it would probably do no harm, it is quite unlikely that it would do any good. It could have tragic consequences if it were employed in chronic otitis media of Lillie's[2] type III or type IV, in which surgery is clearly indicated.

As I related in my first communication on this subject,[1] my attention was attracted to the possible usefulness of sulfamylon in otitic infections by Clark's report on its effectiveness in the prevention of infection after ophthalmic surgery. His published report[3] concerned 84 successive operations after which Bacillus pyocyaneus, formerly an extremely troublesome secondary micro-organism, was completely controlled by the use of sulfamylon in 1 per cent solution for irrigations and moist dressings. Although dressings had to be changed frequently, sometimes for as long as a month after operation, there was not a single instance in the series of any of the reactions experienced under similar circumstances with other sulfonamide preparations.

BACTERIOLOGIC STUDIES.

Sulfamylon, which is derived from benzalamine, differs from sulfanilamide in that a methylene group has been inserted between the benzene ring and the 4-amino group. It is available as a crystalline white substance which is freely soluble in water.

Sulfamylon has the dual advantage of being both effective and innocuous. Its antibacterial activity, Brewer[4] demonstrated, is not inhibited by p-aminobenzoic acid and, therefore, its range of usefulness is wider than that of other sulfa compounds. Brewer's additional studies, which were confirmed by Lawrence,[5] showed that sulfamylon is more effective against aerobes and anaerobes *in vitro* than are comparable

concentrations of sulfanilamide and sulfathiazole. Still other investigations showed that solutions ranging in strength from 0.5 to 2 per cent did not harm the conjunctiva of rabbits and that solutions ranging in strength from 1 to 20 per cent did not harm the bladder mucosa. Comparative studies by Howes[6] showed that for local application sulfamylon has a wider range of usefulness than streptomycin, penicillin, parachlorophenol, tyrothricin or zepharin. Its special advantages, as this observer listed them, are that it is rapidly bactericidal, it is not affected by changes in the acidity of the environment, it is active in the presence of pus and blood, and it is relatively nontoxic.

Studies made for me in the laboratories of Our Lady of the Lake Sanitarium in Baton Rouge showed that sulfamylon was consistently effective *in vitro* against the organisms commonly found in otitis externa and chronic otitis media, that is, pseudomonas aeruginosa, staphylococcus aureus, beta hemolytic streptococcus, Klebsiella pneumoniae, alpha hemolytic streptococcus and escherichia coli. Solutions of sulfamylon ranging in strength from 4 per cent down to 0.25 per cent effectively inhibited growth in all of the 36 cultures tested. Additional studies showed that the optimum bactericidal effect was obtained when a 1 per cent solution was kept in contact with the bacteria tested for a five-minute period. This is a useful and practical bit of information: The success of any mode of treatment of otitic infections depends upon the cooperation of the patient and most patients who are not incapacitated by their condition will not submit to inactivity for much more than five minutes at a time.

ROUTINE OF MANAGEMENT.

The present routine of management in otitis externa, chronic otitis media and occasional postoperative infections has been somewhat modified from the routine described in my first communication.[1] I am in full agreement with Fowler[7] that bacteriologic studies are essential in these conditions, but I am in equally full agreement with Senturia[8] that for a variety of reasons they are not uniformly practical in private

practice. I should not advise anyone who is just beginning to treat otitis externa and allied conditions to omit them. On the other hand, I am convinced from my own experience that as experience increases, it is frequently possible to determine from inspection and clinical examination what sort of infection one is dealing with and that it is entirely justifiable to treat most patients tentatively, on the basis of that impression, with a considerable saving of time and expense. If by the second visit the ear has not shown great improvement under the treatment instituted, cultures are taken at once. In this connection might be mentioned Senturia's recent statement, in his discussion of Fowler's[7] paper on topical applications to the external ear, that the Los Angeles Research Study Club was sponsoring an investigation which, it was hoped, would produce a method of cytologic examination which could be carried out in the office to differentiate among the varieties of external otitis. It would be an enormous diagnostic aid if this could be achieved.

The present routine of treatment is as follows:

1. Following history-taking and the usual general and special examinations, a smear and culture are taken if there is any doubt at all concerning the type of infection presented. The ear is then thoroughly cleansed with the tip suction, at first with the smallest size Frazier suction tip, then, near the attic and the tympanic membrane, with a tip devised in my office for this purpose. This tip is malleable and can be bent to reach otherwise inaccessible areas. Finally, the canal is irrigated with one ounce of 95 per cent alcohol and is thoroughly dried.

2. If the infection is thought to be fungoid in origin, the fungicide of choice is prescribed. I prefer thymol and cresatin for this purpose. Otherwise, sulfamylon is applied and the patient is shown how to use it at home. Application is simple: The patient lies on the nonaffected side, instills the sulfamylon solution into the ear and permits it to remain for exactly five minutes by the clock. The ear is then blotted dry

with cotton and a cotton wick is inserted, which is changed often enough to prevent its becoming saturated with secretions from the ear.

3. When the patient returns to the office, usually 48 hours after the first visit, the ear, as a rule, is found greatly improved, probably as the result of the thorough cleansing combined with the action of the sulfamylon. My own belief is that if it were practical for patients to come to the office daily, it would be possible to clear up most cases of otitis externa merely by repeated daily cleansing. It is of interest that almost without exception observers who report good results with various methods of treatment all emphasize the importance of cleansing the canal and drying it thoroughly before any applications are made to it.

If on the second visit the infection is clearly under control, the ear is again cleansed and dried carefully and is dusted with sulfanilamide powder. The patient is instructed to discontinue all medication and to make every effort to keep water out of the ear for three to four weeks. If, however, improvement has not occurred, the initial treatment is repeated and the patient returns to the office at regular intervals, preferably 48-hour intervals, until control is achieved.

4. In cases in which a culture has been made on the first visit or on a subsequent visit (as is routine in cases in which improvement is not promptly apparent), treatment depends upon the results of laboratory examinations. The report usually shows *1.* mixed organisms, with pseudomonas predominating, *2.* mixed organisms with staphylococcus predominating, *3.* streptococcus, with the precise type named, or *4.* B. pyocyaneus. If a pure staphlococcus is reported, or a mixed infection with staphylococcus predominating, a coagulase test is run. If this test is reported negative, the staphylococcus is disregarded and the infection is treated as if it were gram-negative. That is, sulfamylon therapy is continued. If the test is reported positive, attention is directed toward the staphylococcus. Sulfamylon therapy is discontinued and the patient

is treated with penicillin intramuscularly and locally (by drops of a solution containing 100,000 units of penicillin per ounce of distilled water), or by furacin.

To reiterate the principles upon which this clinical routine is based: *1.* The drug selected is applicable to the individual case. *2.* Whatever agent is used is applied in a high enough concentration and over a sufficient period of time to control the infection. *3.* Before any application is made to the ear, and as an essential part of the treatment, the ear is carefully cleansed and dried. The drug is thus enabled to reach all parts of the ear and the aural secretion, the pH of which might alter the effectiveness of the drug, is eliminated, at least for the time being. *4.* The drug is applied only long enough to achieve the desired effects. The long continued use of any type of topical application may induce sensitivity. Dermatologists are of the opinion that penicillin cannot be used locally much longer than about five days without the appearance of erythema, vesiculation and other evidences of sensitization. My own feeling is that similar time limits might well be set upon all topical applications. *5.* If bacteriologic studies are made and a gram-positive organism is found, sulfamylon is withheld, for use in the event that the strain proves resistant, and some other form of treatment is employed first.

ANALYSIS OF CASES.

My preliminary report on the use of sulfamylon in external otitis and other otitic infections concerned 141 infections in 108 patients. In the series were 33 cases of external otitis, 24 of chronic otitis media, nine infections secondary to the fenestration operation and seven secondary to tympanomastoidectomy. The 73 cultures made revealed pseudomonas aeruginosa, staphylococcus aureus, beta and alpha hemolytic streptococcus, Klebsiella pneumoniae, and escherichia coli. Sulfamylon was effective in every instance in this series. In 93 cases the infection was controlled by the seventh day and in no case was treatment necessary longer than 14 days. The single reaction in the series was proved to be the result of a local allergy which cleared promptly after sulfamylon was discontinued and an antihistaminic drug was administered.

To my first series of sulfamylon-treated otitic infections I am now adding a second series of 164 infections in 122 patients. Sixty-five patients had otitis externa, 34 chronic otitis media, 12 cavities following tympanomastoidectomy, and 11 cavities following the fenestration operation. The bacteriologic results were in general agreement with those in the first series.

The results of treatment were quite as satisfactory as in the first series. In 80 cases the infection cleared up within two days, in 24 within four days, in 12 within seven days, in three within nine days, and in three within 11 days. The rapidly favorable results — in this second series only six patients paid more than three visits to the office — are advantages which commend this method to physician and patient alike. There were no reactions at all in the second series.

It is true that I have made no formal follow-up of these 230 patients, but I am sure that most of you will understand me when I say that although my practice is exclusively limited to a specialty, it is more or less of the family type, as might be expected in a small city. I have seen most of these patients for other conditions, or heard of them through other patients who were relatives or friends, or have met them as I have gone about my affairs. I feel sure that in the two series there were some recurrences of infection, but I feel equally sure that most of the patients treated were permanently relieved.

COMMENT.

Reports on the use of sulfamylon in otolaryngology are still few. Fox,[9] in 1947, treated 137 patients with rhinosinusitis by this means with such good results that he concluded that this drug was "the most valuable of all chemotherapeutic agents in the local treatment of acute rhinitis and sinusitis." In most cases in the series no other treatment was used, and antrostomy, which had been Fox's practice if antral infections did not clear up after three or four irrigations, was not required in a single case.

The only report of which I am aware which concerns the use of sulfamylon in otitis conditions is by Fowler,[7] in 1948. He used this agent in the treatment of both otitis externa and chronic otitis media whenever pseudomonas was found in the culture. In the first of the 17 cases in his series he applied the drug in 5 per cent concentration in a water-soluble ointment base (carbowax). In later cases he used a 1 per cent solution of methyl cellulose, with a pH adjusted from 4.5 to 6.5.

Diehl and Morris'[10] report deals with a group of patients with otitis media treated with sulfamylon and streptomycin. Fowler[7] used the same combination for mastoid cavities. The addition of streptomycin makes it impossible to evaluate either series from the standpoint of the effectiveness of sulfamylon alone.

I would not, of course, wish to indicate that sulfamylon is the only possible method of treatment in otitis externa and chronic otitis media. There are several other useful methods, in addition to penicillin therapy. My personal experience with dibromsalicylaldehyde (compound 280) was entirely unfavorable and did not in any way duplicate the results reported by Hayes and Hall.[11] I have used furacin, however, with very satisfactory results in coagulase-positive staphylococcic infections, my experience being in accord with the results reported for this form of treatment by Anderson and Steele[12] and by Douglass.[13]

SUMMARY AND CONCLUSIONS.

1. The situation in respect to the management of otitis externa, chronic otitis media, and certain postoperative otitis infections is greatly improved over what it was.

2. Experience suggests that if the ears are carefully cleansed, if care is taken to determine the causative organisms and to use therapeutic agents to which they are sensitive, much of the confusion surrounding the treatment of these conditions can be expected to disappear.

3. It is the duty of otolaryngologists, as Senturia[8] has emphasized, to continue their methods and techniques critically and to confine their therapy to the use of therapeutic agents worthy of trial.

4. This report concerns the treatment with sulfamylon of 164 infections in 122 patients, which are added to the previously reported series of 141 infections in 108 patients. In both series the majority of cases were instances of external otitis and chronic otitis media, but both included a few instances of infection following tympanomastoidectomy and the fenestration operation. In both series the response to sulfamylon therapy, combined with thorough cleansing of the ear, was uniformly satisfactory and almost uniformly rapid. There was one reaction, as the result of local allergy, in the first series but no reactions at all in the second.

REFERENCES.

1. McLaurin, J. W.: The Local Application of "Sulfamylon" [Para-(Aminoethyl)-Benzene Sulfonamide Hydrochloride] in Otitis Externa and Chronic Otitis Media. A Preliminary Report of 141 Infections in 108 Patients. The Laryngoscope, 58:1201, 1948.

2. Lillie, Harold I.: An Empirical Clinical Classification of Chronic Suppurative Otitis Media. *Atlantic Med. Jour.*, 31:559, 1928.

3. Clark, William B.: Local Use of "Sulfamylon" [Para-(Aminoethyl)-Benzene Sulfonamide Hydrochloride]. *Arch. Ophth.*, 38:682, 1947.

4. Brewer, J. H.: Clear Liquid Mediums for the "Aerobic" Cultivation of Anaerobes. *Jour. A. M. A.*, 115:598, 1940.

5. Lawrence, C. A.: *In Vitro* Studies on the Antibacterial Actions of Para-Aminoethylbenzenesulfonamide Derivatives. *Jour. Bact.*, 49:149, 1945.

6. Howes, E. L.: Local Chemotherapy of Wounds. *Surg., Gynec. and Obst.*, 83:1, 1946.

7. Fowler, Edmund P.: Topical Applications to the Skin of the Ear Canal. *Tran. Amer. Acad. Ophthal.*, p. 637, 1948.

8. Senturia, Ben H.: In discussion of Fowler.[7]

9. Fox, Samuel L.: The Use of Sulfamylon in Rhinosinusitis. *Ann. Otol., Rhinol. and Laryngol.*, 56:946, 1947.

10. Diehl, Kenneth L., and Morris, Austin G.: Sulfamylon and Streptomycin in Treatment of Infections of the Ear. A Preliminary Report. *Arch. Otolaryngol.*, 50:700, 1949.

11. Hayes, Merrill B., and Hall, C. Fremont: The Management of Otogenic Infection. *Tran. Amer. Acad. Ophth.*, p. 149, 1947.

12. Anderson, Jack R., and Steele, C. H.: Use of Nitrofuran Therapy in External Otitis. The Laryngoscope, 58:1279, 1948.

13. Douglass, Carleton C.: The Use of Furacin in the Treatment of Aural Infections. The Laryngoscope, 58:1274, 1948.

THE LYMPH PUMP MECHANISM OF THE NOSE AND PARANASAL SINUSES.*†‡

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The lymph pump mechanism of the nose and paranasal sinuses essentially consists of the anatomic and physiologic adaptation of the sinal mucosa and turbinal tissue to the process of adjusting their tissue pressure to a magnitude which promotes the flow of lymph from their tissue spaces. Other interrelated variables which enter into this mechanism, particularly during pathologic states, are:

1. The relation of the normal and pathologic tissue pressure of the turbinal tissue to the tissue pressure of the sinal mucosa.

2. Lymphatic blockage in the sinal mucosa or in the retropharyngeal lymph nodes.

3. The relation of the colloidal osmotic pressure of the sinal secretion to the colloidal osmotic pressure of the interstitial fluid in the sinal mucosa.

4. The presence of relatively unremovable protein or bacterial products or antigens in the turbinal tissue or sinal mucosa. This factor is important during both purulent and allergic states.

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Information concerning this mechanism has been derived from the following sources:

1. Clinical observation of the relation of nasal stenosis due to increased turgescence of the turbinates to sinal pathology occurring during various types of rhinitis and sinusitis.

2. Study of the histology and pathology of the sinal mucosa with particular reference to its anatomical, physical and functional characteristics.

3. Determination of the colloidal osmotic pressure of the sinal secretion and interstitial fluid in certain types of sinusitis. Examination of these fluids has had the following obvious limitations:

Sinal secretion and interstitial fluid must be present in sufficient quantity and procurable before they can be examined. During acute purulent sinusitis secretion is plentiful and easily obtained and often, as the sinal mucosa becomes very edematous, the interstitial fluid may be aspirated; however, during subacute allergic sinusitis usually it is impossible to obtain secretion or interstitial fluid from the sinal mucosa. Also during chronic purulent sinusitis, although secretion is readily available, the interstitial spaces have become so filled with round cells or fibroblasts that interstitial fluid is not procurable. In instances where specimens are not procurable, opinions of the processes are based upon observations of similar pathology occurring elsewhere, particularly elephantiasis.

The osmotic pressure of the specimens of secretion and tissue fluid examined has been calculated by determining the percentage of albumin and the total protein and using the nomogram of Wells, *et al.*, for computation. The actual numerical values of the protein, osmotic pressure, etc., are not reported in this paper. They will be presented as part of a more technical article.

The physiologic need for the lymph pump mechanism is derived from the requirement of every living cell for food and an excretory mechanism proportioned to the cells' activity. In mammals these requirements are provided by an

extravascular tissue fluid derived from the blood plasma which produces a constantly changing pericellular environment for the cells of the tissues. The filtrate derived from the arterial capillary contains water, salts and food (protein) for the body cells. The water, salts and certain diffusable products are absorbed from the extravascular fluid by the venous capillary, leaving the products of cell metabolism, particulate matter, cellular detritus, bacterial products and unused protein to be carried away by the lymphatic system. This arrangement is, in effect, a filtering process which allows molecules of various sizes to find their way from the interstitial spaces to the blood stream by different routes with little or great delay or difficulty. Mammalian lymph flows normally by muscle contraction, massage, active and passive motion of the body, gravity, tissue pressure and pulsation transmitted from the arteries to the lymph vessels. During pathologic states, when the part is immobile, lymph flows by pathologic or increased tissue pressure, gravity and pulsations transmitted from the arteries and dilated arteriovenous anastomoses to the lymph vessels.[1]

In the normal nose and sinuses the tissues are deprived of muscle contraction, active and passive motion and massage to promote lymph flow. Instead, these structures depend upon gravity and normal tissue pressure. When irritation or infection occurs in the nose and sinuses, lymph flow is accelerated by increased or pathologic tissue pressure, gravity and pulsation transmitted from the arteries and dilated arteriovenous anastomoses to the lymph vessels. This limitation of measures available for moving the extravascular fluids in the tissues of the nose and sinuses is reflected in their anatomy. The anatomic adaptation of these structures to their physiologic needs is evident in the molding of the turbinal tissue to irregularities of the nasal cavities and the physical characteristics of the sinal mucosa and turbinates which allows them to alter their tissue pressure rapidly by swelling against their inherent elasticity and contractility and their bony enclosure.

Tissue pressure is gauged by the resistance a tissue offers to dilatation of its interstitial spaces by edema fluid. Accord-

ing to its magnitude, tissue pressure offers resistance to capillary filtration and accumulation of an excessive amount of extravascular fluid, and promotes capillary absorption and lymph flow. Tissue pressure is proportional to the density and elasticity of the tissue, increases with the rapidity of capillary filtration, and becomes less as the elasticity of the tissue is lost by long continued distention of its interstitial spaces by edema fluid.[1] An increase in tissue pressure is resisted by the fascial planes, integument covering the part, and bony enclosures of the body. Tissue pressure may be increased by application of physical pressure to the surface of the part. Other pressure variables which affect the origin, distribution and absorption of extravascular fluid are arterial and venous capillary blood pressure, osmotic pressure, hydrostatic pressure and atmospheric pressure.

Anatomically the lymph pump mechanism may be divided into two parts: one being the lymph bed of the nose and paranasal sinuses, and the other the retropharyngeal lymph nodes into which the lymph bed drains. Each of these parts when involved in pathologic states manifest their functional pathology by clinical signs and symptoms which are of diagnostic value.

The retropharyngeal nodes are from two to 10 in number during childhood. These are divided into medial and lateral groups. The medial group atrophies during adulthood, usually leaving only one of the lateral retropharyngeal nodes on one side. Through these nodes drains all of the lymph from the structures developed from the first branchial pouch, *i.e.*, the middle ear, Eustachian tube, adenoid, nasopharynx, nasal cavities and paranasal sinuses. Lymph from these structures passes through the lymph capillaries and pathways in the mucosa of the nose and nasopharynx into either pretubal lymphatic plexus. Efferent vessels from these plexuses pierce the pharyngeal aponeurosis and enter the retropharyngeal nodes. The pretubal lymphatic plexuses anastomose freely through channels which traverse the pharyngeal surface of the soft palate. This free communication between the pretubal plexuses is important to an understanding of the bilat-

eral character of lymph edema occurring during acute blockage of the retropharyngeal nodes and the retrograde metastasis of cancer cells in this region; also, these channels may be the route of flow from one nasal cavity to the other of the increased amount of extravascular fluid present in the tissues during certain pathologic states.[3]

The physic qualities and anatomic structure of the sinal mucosa and turbinal tissue are specifically designed and adapted for the purpose of promoting lymph flow from the stroma of the mucosa and drainage of sinal secretion. To understand better this adaptation, it is important to recall that all air-containing cavities of the body, when they become severely infected, tend to lose their air by becoming filled with edematous tissue and/or secretion. As air is driven from a cavity the tissue pressure of the tissues in the cavity is raised and pulsations from the arteries and dilated arteriovenous anatomoses are transmitted to the lymph vessels through an incompressible fluid medium and thus lymph is pumped toward the lymph nodes. The perfection of this process usually is related to or varies with the virulence and invasive characteristics of the offending agent.

To facilitate discussion of the adaptation of the lymph bed to the function of promoting lymph flow, it is useful to divide it into two functionally related parts; one being the sinal mucosa and the other the turbinal tissue. The normal sinal mucosa is "seldom more than 0.02 mm. in thickness"[4] and consists of a very thin stroma of closely placed fibroblasts surmounted by the pseudostratified ciliated epithelium. On section the cells of the stroma appear to be placed so close to each other that there are relatively few intercellular or interstitial spaces present. When the stroma of the normal mucosa is teased apart, the cells separate into layers with the lines of cleavage appearing parallel to the epithelial surface. This sparse cellular and tightly layered construction of the stroma of the normal sinal mucosa accounts for the facility with which the mucosa becomes edematous, approximates the capacity of the sinus and thereby raises its tissue pressure. Also, the scarcity of interstitial spaces and blood vessels in

the normal sinal mucosa precludes absorption from its surface. Some form of conditioning process which dilates its interstitial spaces and provides more blood supply is necessary before absorption can occur through this membrane. Conditioning of this membrane for absorption will be considered under allergic sinusitis.

Drainage from the normal sinus is accomplished chiefly by ciliary action. During pathologic states drainage is accelerated by reduction in the size of the sinal cavity by swelling of the sinal mucosa. During inflammation the degree of swelling of the sinal mucosa is greater in that part of the mucosa which is far removed from the ostium of the sinus and swelling of the mucosa is least in the region of the ostium. This provides for drainage of the sinal cavity by the edematous mucosa forcing the secretion to or through the ostium of the sinus. When swelling of the mucosa becomes extreme, portions of the mucosa may prolapse through the ostium as polypoid protrusions.

It is important to an understanding of this factor in sinal drainage to consider the relation of the anatomic structure of the sinal mucosa to its tissue pressure. The opinions expressed here have been derived chiefly from studies of the mucosa of the maxillary sinus. Sections of the mucosa from various parts of this sinus show it to contain more glands, blood vessels and lymph vessels near the ostium. Also, it is thicker and more dense in the area of the ostium than it is in areas far removed from the ostium. Since tissue pressure resists capillary filtration and is related to the density and elasticity of tissue, it is evident that following a given uniform stimulus to increased capillary filtration, the filtrate appears first and dilates to a greater degree the interstitial spaces in that portion of the sinal mucosa which are away from the ostium of the sinus. As the edema of the sinal mucosa progresses, the mucosa displaces the air from the sinal cavity and forces the secretion toward the region of the ostium of the sinus. During this process the ostium remains patent, due to the relatively greater tissue pressure of its mucosa.

The variables which affect capillary filtration are the colloidal osmotic pressure of the blood, the permeability and blood pressure of the arterial and venous capillaries and the tissue pressure. If other factors remain constant and there is lowering of the colloidal osmotic pressure of the blood as in simple hypoproteinemia, increased capillary filtration occurs first into areas of the body which have the least tissue pressure. This is commonly seen clinically in the edema in the loose tissue around the eyes during hypoproteinemia accompanying albuminuria, and in the feet and ankles where hydrostatic pressure and gravity play a part in increasing capillary filtration and collection of fluid in the interstitial spaces. Gravity also may play a part in the distribution of edema fluid in the sinal mucosa. If the sinal mucosa is very edematous the fluid may become relatively free, *i.e.*, not a part of the tissue gel. As tissue fluid becomes free to move in the interstitial spaces of the sinal mucosa it gravitates to the most dependent portion of the sinus. Shifting by gravity of this edema fluid to the most dependent part of the sinus also is a factor in bringing secretion to the level of the region of the ostium and facilitates sinal drainage. This clotting tissue fluid often may be aspirated from the sinal mucosa through a needle but should be differentiated from similar appearing nonclotting cystic fluid or the fluid portion of the secretion contained in the sinal cavity.

In cadavers with generalized edema and otherwise normal sinuses which have undergone rigor mortis in dorsal decubitus, the mucosa of the posterior walls of the sinuses is much more edematous than the mucosa on the other walls. There is some evidence that the tissue pressure of the sinal mucosa is probably less than that of any other tissue of the body. If this be true during severe hypoproteinemia the sinal mucosa is the first tissue to become edematous and the last tissue from which the edema fluid is absorbed. During X-ray examination using contrast media, this type of edema has been interpreted erroneously as primary sinal pathology.

The intranasal structures aid in changing the extravascular fluid and promoting lymph flow by the same fundamental

methods utilized by the sinal mucosa. These structures differ from the sinal mucosa anatomically and physiologically in that the nasal mucosa has greater normal tissue density and higher normal tissue pressure, while the turbinal tissue in addition to these qualities possesses inherent erectility and elasticity and its smooth muscle fibres provide for more enduring contractility.

All of the lymph from the mucosa of the sinuses must pass through the lymph channels in the nose en route to the retropharyngeal nodes. In the normal nose the tissue pressure of the turbinates and nasal mucosa is so balanced against capillary filtration that only enough fluid and protein is filtered into the tissues to meet the physiologic requirements; therefore, the intranasal tissue is able to remove the nomal extravascular fluid without abnormal turgescence. Following irritation or infection of the nasal mucosa, there is an increase in the amount of interstitial fluid to be removed from the lymph bed, so the intranasal structures drive air from the nasal cavity by swelling against their surrounding bony walls. The lymph can then be pumped from the lymph bed by pulsations transmitted from the arteries to the lymph vessels through an incompressible fluid medium.

The anatomy and physiology of the normal nose provide for nasal cavities either of which alone can transmit sufficient air to the lungs for normal, quiet respiration while the opposite cavity is closed. During moderate irritation or infection, swelling of the turbinal tissue alternates from one nasal cavity to the other, *i.e.*, the right nasal cavity will close while respiration occurs through the left, then the left will close and respiration occurs through the right. This alternating cycle of opening and closing of the nasal cavities was described by the Hindus during the Vedic Period (1500 B.C.). A British anatomist, Hemchandra Sen, in 1901, described how one and then the other of his own nasal cavities would alternately open and close every few hours. His observations were made by sticking his tongue up behind his soft palate and examining the condition of his turbinates, noting that one side of his nose would close, alternating with the other side.[5]

The alternate opening and closing of the nasal cavities may be interrupted by gravity. In lateral decubitus, the nasal cavity which is inferior will close, while in dorsal decubitus both nasal cavities will remain open when there is only a moderate amount of extravascular fluid to be removed from the lymph bed. When gravity fails to affect or aggravates intranasal tissue turgescence, usually the reaction to the agent which caused the intranasal swelling has been severe. Also, gravity may have little effect during partial or complete lymphatic blockage of the retropharyngeal lymph nodes, or when the protein content and colloidal osmotic pressure of the edema fluid in the sinal mucosa is very high.

The presence, location, cytology and physical characteristics of nasal and sinal secretion, the color and swelling of the sinal mucosa, and the anatomic construction of the nasal cavities are diagnostic criteria important during rhinitis and sinusitis; however, the degree and location of swelling or turgescence of the turbinal tissue also is a most valuable diagnostic aid in nasal and sinal disease.

The functional pathology of the turbinates consists of their becoming turgescent and increasing their tissue pressure as an auxiliary or aid to the sinal mucosa as the mucosa for functional purposes needs to increase its tissue pressure.

The character and degree of turgescence of the turbinates are related to the type and extent of lymphatic blockage present in the sinal mucosa. Also, the tissue pressure of the sinal mucosa, the colloidal osmotic pressure of the secretion and interstitial fluid of the sinal mucosa, and the presence in the tissue spaces of the sinal mucosa of relatively unremovable substances are important factors affecting turgescence of the turbinates. During purulent infections these relatively unremovable substances, until more information concerning their true nature is available, may be regarded as plasma colloids and bacterial products, while in allergic states they may be regarded as air- or blood-borne protein antigens. If the allergic state is long continued, bacterial antigens from secondary infection also may be present. During acute purulent states

the sinal mucosa becomes edematous and increases its tissue pressure in direct proportion to the colloidal osmotic pressure of its edema fluid. If the colloidal osmotic pressure of the secretion in the sinus is high and holds in the stroma of the mucosa fluid of high protein content the turbinates will remain turgescent until the secretion is drained from the sinus and the colloidal osmotic pressure of the edema fluid in the sinal mucosa is reduced. As the protein in the edema fluid is carried away, there is no longer need for an accessory swelling of the turbinates to increase the tissue pressure as an aid to removal of components of the extravascular fluid. To understand the interrelationship of these variables it is important to consider their relation to lymph flow.

Lymphatic blockage in the mucosa of the lymph bed is the principal factor in determining the relation of turgescence of the turbinates to pathology in the sinuses. It may be recognized in three stages: acute, subacute and chronic. Hindrance to lymph flow in either of these stages may be partial or complete. Acute lymphatic blockage occurs as fibrin, cellular detritus, and round cells infiltrate the intercellular and interstitial spaces of the sinal mucosa. During this stage blockage of the interstitial spaces is usually so complete that nothing except certain diffusable toxic products pass through the walled off area. This results in retention in the mucosa of a highly proteinized tissue fluid rich in albumin with correspondingly high colloidal osmotic pressure. During this stage the turbinal tissue of the same side is constantly and completely turgescent, or as alternating nasal stenosis ensues the turbinates on one or both sides may recede incompletely during the cycle of alternation.

During the subacute stage of lymphatic blockage the fibrin and cellular detritus have been absorbed, leaving only the round cell infiltration. In this stage of lymphatic blockage the interstitial spaces in the stroma of the sinal mucosa are so filled with round cells that there is little room for excess extravascular fluid. The turbinates during this stage may appear normal or show alternating turgescence during acute exacerbation of infection. Subacute lymphatic blockage may

continue for years without seriously interfering with nutrition of the mucosa. This type of mucosa may be returned to a semblance of normal by application of physical pressure to its surface.[6] During chronic lymphatic blockage in the sinal mucosa, fibrosis of the stroma of the mucosa is present and there are relatively few interstitial spaces to hold fluid. This type of blockage usually terminates in loss of nutrition to the glands and ciliated epithelium of the mucosa. Often this type of mucosa is seen surrounding a septic cavity. The turbinal tissue in this stage is not turgescent because there is little extravascular fluid to be removed from the stroma of the sinal mucosa. Also, the pathways of exit for lymph from the sinal mucosa are so blocked by fibrous tissue that there is no stimulation to the turbinates to increase their tissue pressure by becoming turgescent.

The clinical application of the foregoing principles of nasal physiology and functional pathology will now be applied to certain types of nasal stenosis due to turgescence of the turbinates. Turgescence of the turbinates will be classified as alternating, when it changes from one nasal cavity to the other, and as intermittent, constant or complete when either or both nasal cavities are closed intermittently, constantly or completely.

I. Excessive Intranasal Medication. Patients suffering from this frequently self-administered condition usually begin with mild bacterial or allergic rhinitis and alternating nasal stenosis. They usually give a history of having administered "nose drops" or inhalations at increasingly frequent intervals until they have bilateral constant, complete nasal stenosis. These patients have overstimulated the contractile power of their turbinal tissue, and must stop their medication and necessarily go through a period of constant nasal stenosis until their turbinates regain their contractility. In this condition it is important to impress on the patient that during certain pathologic states it is necessary for the nasal cavities to become blocked as part of their functional pathology before they can return to normal; therefore, if they continue their intranasal medication they only postpone the time when the block-

age will occur. Some of these patients are aided greatly by irrigation of their maxillary sinuses and evacuation of trapped secretion.

II. Nasal diphtheria and glanders produce acute lymphatic blockage of the retropharyngeal lymph nodes and cause constant complete nasal stenosis due to lymph edema of the lymph bed. Frequently this type of swelling of the intranasal tissues cannot be reduced with adrenalin, cocaine or other commonly used shrinking agents.

III. Acute purulent sinusitis is recognizable clinically in three stages:

1. Empyema of the Sinus. In this stage the sinus contains secretion having a protein content frequently as high or higher than the protein of the blood plasma with a corresponding high colloidal osmotic pressure. This high osmotic pressure secretion holds in the stroma of the sinal mucosa interstitial fluid high in protein. This is reflected intranasally in marked turgescence of the turbinates on the affected side as the lymph pump mechanism attempts to remove the highly proteinized tissue fluid. The turgescence of the turbinates in this stage is due to marked dilatation of their cavernous bodies, lymph edema of the intranasal mucosa and blood vessel engorgement. As lymphatic blockage in the sinal mucosa becomes complete the turgescence of the turbinates may subside slightly.

If the sinusitis is unilateral the nasal stenosis then may become unilaterally intermittent, or if it is bilateral it may alternate from one nasal cavity to the other as the process passes into the draining stage.

2. Draining Stage. In this stage the colloidal osmotic pressure of the sinal secretion has fallen below that of the edema fluid in the sinal mucosa. This allows the colloids to be removed from the edema fluid in the sinal mucosa by the lymphatic system and the water and electrolytes to be absorbed by the veins. During this stage, if the sinusitis is unilateral the nasal stenosis due to turgescence of the turbinates is intermittent on the affected side. If the sinusitis is

bilateral, alternating nasal stenosis occurs, excepting when the turbinates are affected by gravity. The effect of gravity in this stage is evidenced clinically by placing the patient with normally constructed nasal cavities in lateral decubitus and noting that the nasal cavity uppermost is open while the inferior cavity is closed by turgescence of the turbinates. Also, if the patient is placed in dorsal decubitus, both nasal cavities will open unless there is partial or complete lymphatic blockage of the retropharyngeal lymph nodes. There is no exit from the nose anteriorly for lymph or excess extravascular fluid; therefore, if the patient is placed in ventral decubitus, both nasal cavities may close if the sinusitis is bilateral. Factors such as toxins, tissue destruction and specific invasive characteristics of certain organisms may alter these clinical findings.

3. Resolution Stage. The sinal secretion has almost disappeared in this stage and has become definitely separately into its clumped and fluid portions. The osmotic pressure of the secretion is so low it is negligible, and the sinal mucosa has become sufficiently edematous to increase its tissue pressure to a magnitude which promotes lymph flow. Often the mucosa is so edematous it completely fills the sinal cavity. When this occurs in the maxillary sinus and the sinus is punctured with a needle under the inferior turbinate, the needle may enter the edematous mucosa and as irrigation is attempted part of the edematous mucosa may be forced against the ostium and block the irrigation. Clinically it is important to understand that this marked swelling of the sinal mucosa is an essential part of its functional pathology. In the stage of resolution, even though the sinal mucosa is very edematous, if the mucosa is not mistreated it will rapidly return to normal. The alternating or intermittent nasal stenosis which ushers in the stage of resolution gradually subsides until the turbinates return to normal.

IV. Chronic Purulent Sinusitis. The protein content and osmotic pressure of the sinal secretion during chronic purulent sinusitis are high, but the interstitial tissue spaces of the sinal mucosa have become filled first with round cells and

later with fibroblasts which have blocked the lymph pathways. This type of mucosa contains little protein that can be removed by the lymph pump mechanism; therefore, there is no increased turgescence of the turbinates or nasal stenosis to betray the presence of purulent secretion in the sinus.

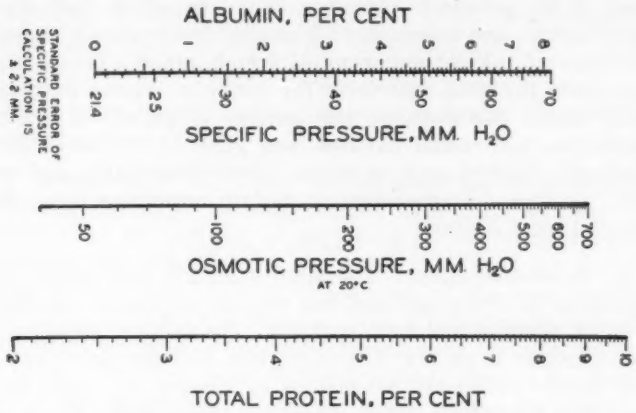

Fig. 1. "Nomogram. A straight line drawn through the proper points on the albumin and total protein scales will intersect the middle scale at the value of the total osmotic pressure calculated from the formula, $P = C(21.4 + 5.9A)$. Where P is the osmotic pressure in millimeters of water, C is the total protein concentration and A is the albumin concentration in grams per 100 cc. The standard error of calculation is ± 5 per cent." (From H. S. Wells, J. B. Youmans and D. G. Miller, Jr., Jour. Clin. Invest., 12:1103, Nov., 1933.)

V. Allergic Rhinitis and Sinusitis. Purulent rhinitis and sinusitis is primarily a disease of the surface of the mucosa, while allergic rhinitis and sinusitis primarily affects the stroma of the mucosa. These two conditions also differ in their stimulation to increased permeability of the capillaries. During purulent disease the increased permeability of the capillaries allows a relatively large quantity of protein to filter into the tissues, while during allergic disease, although the edema may occur very suddenly and extensively, the protein content of the capillary filtrate is relatively low. Clinically, this is evidenced by the rapidity of absorption, probably prin-

cipally by the veins, of edema occurring during certain allergic states such as urticaria and angioneurotic edema, while the highly proteinized filtrate occurring during purulent disease requires more time for processing and removal of the protein through the lymphatic system. One outstanding difference between purulent and allergic disease of the sinuses is to be found in the relative absence of lymphatic blockage in the lymph bed during pure allergic sinusitis when com-

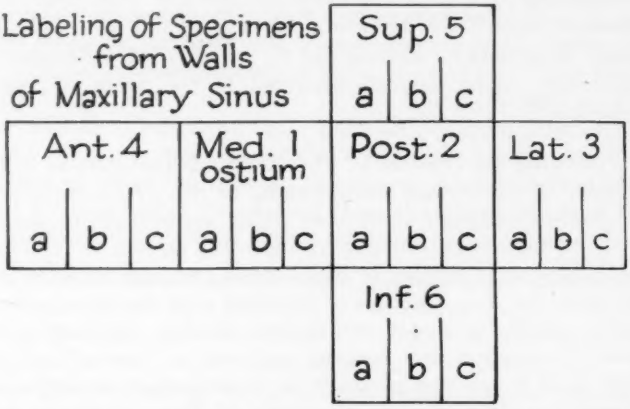

Fig. 2. Method of labeling specimens of mucosa procured from the maxillary sinus for study of normal and pathologic states.

pared to that present during purulent sinusitis. The turbinates become turgescent to aid the sinal mucosa in increasing its tissue pressure; however, the turbinates respond in this manner only if the interstitial tissue spaces in the sinal mucosa between the turbinates and the sinus are "open." During allergic states when relatively unremovable antigenic substances are present in the sinal mucosa the turbinates may manifest increased turgescence constantly until the antigen is removed or until lymphatic blockage occurs in the interstitial spaces of the stroma of the mucosa. Apparently in certain instances the tissue must be conditioned before allergic manifestations can occur in certain areas. Condition-

ing may result from frequent application of an antigen to the same area. Any agent which opens the interstitial spaces of the sinal mucosa to absorption and retention of antigens may act as a conditioning factor. Repeated episodes of purulent or allergic rhinitis may condition the tissue for the occurrence of allergic sinusitis. This is in accord with the Arthus phenomenon which is one of the bases of allergic reactions. "Arthus found that if he injected antigen into animals subcutaneously at definite intervals the first few injections were absorbed readily but later injections were not absorbed. Instead, they became surrounded by an area of inflammation and often tissue necrosis occurred in the areas of recent injection."[7]

Following the reaction of the nasal and sinal mucosa in the manner of the Arthus phenomenon, or one of its variations, the fate of antigenic substances which are brought in contact with its surface or brought by the blood stream to its interstitial spaces is similar to homologous antigens injected into the skin, *i.e.*, one fraction is absorbed into the blood stream either rapidly or slowly and another fraction remains in the area of injection and becomes attached to the cells of the body until it becomes proteolyzed, bacteriolyzed or extruded.[8] Absorption from the sinal mucosa of these antigenic substances of products of their ageing process may produce bronchial asthma in accord with the smooth muscle reaction of Schultz and Dale, or they may produce immunologic shock in accord with the Schwartzman phenomenon. This is in accord with the observations of Landsteiner that "simple chemical substances probably form conjugates with the animal's own proteins, forming new antigens. The presence of these new antigens resulted in sensitization."[7]

1. Acute allergic rhinitis due to inhaled or blood-borne antigens presents alternating nasal stenosis which varies in its severity with the amount of antigen encountered and the sesitivity of the patient. If the allergic reaction is severe or if the patient has indulged in excessive intranasal medication, complete constant nasal stenosis may ensue. Constant com-

plete nasal stenosis occurring during or immediately following the hay fever season often means that the patient has developed acute allergic sinusitis.

2. Acute allergic sinusitis presents constant complete nasal stenosis as the turbinates become turgescent to aid in removing relatively unremovable antigen from the interstitial spaces of the sinal mucosa. Irrigation of the maxillary sinuses in these patients evacuates a small amount of white mucoid secretion containing a marked concentration of eosinophiles. In certain instances particulate matter may be present in the sinal secretion. Irrigation of the maxillary sinuses, preferably by antrum puncture under the inferior turbinate, is of great value in promoting resolution of this type of sinusitis. The clinical improvement of these patients has been much more rapid following irrigation of the maxillary sinus by puncture instead of irrigation through the ostium. This may be due in part to draining of tissue fluid from the fistula made in the edematous sinal mucosa during puncture of the sinus.

The first attack of acute allergic sinusitis is one of the most important episodes in the life of the allergic patient. Often the patient has bronchial asthma for the first time with this type of sinusitis. If the sinus secretion is evacuated promptly after the asthma begins, the asthma usually subsides rapidly; however, if the secretion is allowed to remain in the sinuses the asthma may become protracted and the sinusitis may lapse into the subacute stage.

3. Subacute allergic sinusitis presents a variety of types of nasal stenosis due to turgescence of the turbinates. One complaint most of these patients have in common is alternating or constant nasal stenosis which often is accompanied by symptoms which the patient interprets as "catching cold." The mucosa of the maxillary sinus in this stage may be uniformly edematous and less than 1 mm. in thickness. Apparently the sinal mucosa contains substances which do not stimulate it to increase its tissue pressure sufficiently to promote their removal. In this type of sinusitis the turbinates may show increased turgescence constantly as they attempt to aid the sinal mucosa to increase its tissue pressure.

The subacute stage of allergic sinusitis may last for years and often is accompanied by bronchial asthma which may occur episodically at first and then may become constant. Apparently the sinal mucosa in this stage contains antigen which is undergoing proteolysis, the very slight edema present being a necessary part of this process. X-ray studies of the sinuses, using contrast media, often fail to reveal this slightly edematous mucosa but it can be visualized with an antroscope. This type of sinal mucosa may be returned to a semblance of normal by application of local pressure to its surface.

Frequently the only signs or symptoms presented by the patient during subacute allergic sinusitis are those related to increased turgescence of the turbinates, with or without nasal eosinophilia. Since there is no excess secretion present in the sinuses and the very slight uniform edema of the sinal mucosa is not demonstrable by X-ray, it is easy to overlook the relationship of turgescence of the turbinates to the tissue pressure of the sinal mucosa and erroneously classify many of the early cases of subacute allergic sinusitis as rhinitis due to vasomotor, neurogenic, toxic, endocrine, etc., etiology.

The slight persistent edema of the sinal mucosa present during subacute allergic sinusitis is the first phase of the constantly progressive pathologic changes which are characteristic of allergic disease of the sinuses. The pathologic changes in the mucosa which follow this phase are related to the frequency and severity of secondary infection. Increased turgescence of the turbinates accompanies each episode of secondary infection of the edematous sinal mucosa. Following repeated infection, partial blocking of the interstitial spaces of the stroma of the mucosa by round cells and fibroblasts occurs and stagnation of the edema fluid in the mucosa ensues. This type of mucosa may become so edematous that it may fill entirely the sinal cavity. These pathologic changes usher in the chronic phase of allergic sinusitis.

4. Chronic allergic sinusitis differs pathologically from chronic purulent sinusitis in that the fibrosis which occurs in the stroma of the sinal mucosa during purulent sinusitis is more uniform and complete, while the fibrosis which occurs

in the stroma during chronic allergic sinusitis is irregular and incomplete. This results in the formation of alternating areas of fibrosis and dilated tissue spaces interspersed throughout the stroma of the mucosa. As infection occurs in this type of mucosa and the tissue pressure rises, polypi protrude from the areas of dilated tissue spaces. During this stage the turbinates remain turgescent until lymphatic blockage in the interstitial spaces of the stroma of the sinal mucosa occurs. If lymphatic blockage in the sinal mucosa is incomplete and the sinal cavity is totally or partially filled with polypi, the turbinates may show constant increased turgescence as an aid to removal of protein or bacterial products or antigens from the tissue fluid of the sinal mucosa. Long continued swelling of the turbinates may result in loss of their contractility so that they become boggy and unresponsive to the usual stimuli. As polypi fill the nasal cavities and increase the intranasal tissue pressure the turbinates may become compressed and regain some of their contractility.

SUMMARY.

1. The lymph pump mechanism of the nose and paranasal sinuses essentially consists of the anatomic and physiologic adaptation of the sinal mucosa and turbinal tissue to the process of adjusting their tissue pressure to a magnitude which promotes the flow of lymph from their tissue spaces. Interrelated variables which enter into this mechanism are:

1. The relation of the normal and pathologic tissue pressure of the turbinal tissue to the tissue pressure of the sinal mucosa.

2. The relation of the colloidal osmotic pressure of the sinal secretion to the colloidal osmotic pressure of the interstitial fluid in the sinal mucosa.

3. Lymphatic blockage in the sinal mucosa or in the retropharyngeal lymph nodes.

4. The presence of relatively unremovable protein or bacterial products or antigens in the turbinal tissue or sinal mucosa. This factor is important during both purulent and allergic states.

An understanding of the lymph pump mechanism is useful clinically as an aid in interpreting intranasal findings as they reflect sinal pathology.

The turbinal tissue and sinal mucosa increase their tissue pressure to a magnitude which promotes lymph flow by swelling against their inherent elasticity and contractility and their bony enclosure.

Normally, lymph flows in most of the body by active and passive motion, muscle contraction, massage, gravity, tissue pressure and pulsations transmitted from the arteries to the lymph vessels.

The nose and paranasal sinuses are deprived of active and passive motion and massage to promote lymph flow. Instead, they depend upon tissue pressure, gravity and transmitted arterial pulsations. The latter is important during pathologic states as the tissues swell and obliterate the air-containing cavities so that arterial pulsations can be transmitted to the lymph vessels through an incompressible fluid medium.

The turbinal tissue becomes turgescent and increases its tissue pressure as an auxiliary or aid to the sinal mucosa as it becomes necessary for the sinal mucosa to increase its tissue pressure in an effort to remove substances from the interstitial spaces of the stroma of the sinal mucosa.

Reaction of the turbinates in this manner is governed principally by the freedom with which fluid may move through the interstitial spaces of the nasal and sinal mucosa. During complete blockage of the interstitial spaces in the sinal mucosa to the flow of lymph and extravascular fluid, there is no stimulus to the turbinates to become turgescent. If the interstitial spaces in the sinal mucosa are open and contain relatively unremovable substances the turbinal tissue will remain turgescent until the sinal mucosa has increased its tissue pressure to a magnitude which effects their removal or until these substances become proteolyzed or bacteriolyzed and removed.

Correlation of clinical findings during certain pathologic states with certain principles of physiology and functional pathology of the turbinates and sinuses has been attempted.

BIBLIOGRAPHY.

1. Drinker, C. K., and Yoffey, J. M.: Lymphatics, Lymph and Lymphoid Tissue. Cambridge, Mass.: Harvard University Press, p. 112, 1941.

2. Wells, H. S.; Youmans, J. B.. and Miller, D. G., Jr.: A Formula and Nomogram for the Estimation of the Osmotic Pressure of Colloids from the Albumin and Total Protein Concentrations of Human Blood Sera. *Jour. Clin. Invest.*, 12:1103, 1933.

3. Rouviere, H.: Anatomy of the Human Lymphatic System by M. J. Tobias (translator), New York, N. Y. Ann Arbor, Mich.: Edwards Brothers, 1938.

4. Piersol, G. A.: Normal Histology. Philadelphia, Pa.: J. B. Lippincott Co., 2nd Ed., p. 403, 1894

5. Sen, Hemchandra: Observations on the Alternate Erectility of the Nasal Mucous Membrane. *The Lancet*, 1:564, 1901.

6. Robison, J. Mathews: Pressure Treatment of Allergic Sinusitis. *Arch. Otolaryngol.*, 45:405, Apr., 1947.

Ibid.: Application of Local Pressure to the Mucosa of the Maxillary Sinus. *Tran. Amer. Acad. Ophth. and Otolaryngol.*, May-June, 1947.

7. Sherwood, N. P.: Immunology. St. Louis, Mo.: The C. V. Mosby Co., 2nd Ed., p. 332, 1946.

8. Kahn, Reuben L.: Tissue Immunity. Baltimore, Md.: Charles C. Thomas, p. 529, 1936.

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UNIVERSITY OF ILLINOIS POSTGRADUATE COURSE.

The University of Illinois College of Medicine, Department of Otolaryngology, announces its annual postgraduate course in Basic Otolaryngology beginning Oct. 2, 1950. A full time program of instruction is followed for those intending to prepare for special practice. The course terminates on June 15, 1951. For information and application blanks, contact the University of Illinois College of Medicine, Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

THE USE OF A WETTING AGENT IN LARYNGOTRACHEOBRONCHITIS WITH REPORT OF CASES TO DATE.*

JAMES R. GORMAN, M.D.,
Lynchburg, Va.

We are all faced repeatedly with the terrible problem of laryngotracheobronchitis. Before and after tracheotomy a great many of these cases present severe dryness of the trachea and bronchi, with or without crusting *below* the tracheotomy tube and sometimes with a thick, tenacious secretion which cannot be coughed up by the child.

This past Fall we have had in our area an epidemic of severe proportions, totaling among the pediatricians nearly a hundred cases, three of which required tracheotomies as life-saving measures. Two of them occurred on the same day and the third within a 10-day period. My report has to do primarily with this third case.

CASE REPORT.

Mickey D., white male, 28 months old, was admitted to Virginia Baptist Hospital Sunday, Nov. 6, in a critical condition, requiring immediate tracheotomy; temperature, 102°. Following tracheotomy, the child did fairly well, but the trachea was perfectly dry with very little secretion coming through the tube on coughing, even with the use of normal saline dropped into trachea and suctioned back. Seen at 8:30 A. M., Tuesday, the child was in good condition as to color, breathing and other signs, except dry, severe cough. At 4 P. M. this same day I was called hurriedly to the hospital and found the child in dying condition. The tube was clear, but the child was cyanotic and gasping for air. I thought he would die before we could get him to the operating room, where we had the proper materials, light, suction, etc.

I would like to emphasize one important little point in connection with these dry cases, especially with thick, tenacious discharge and crusts, and that is that I much prefer to use the malleable copper Jackson suction tube in preference to the bronchoscope. I used this tube with suction attached, introduced through the tracheotomy wound, and recovered some very thick, tenacious material which the child was unable to cough

*Read at the combined meeting of the Middle and Southern Sections, American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn. Jan. 17, 1950.

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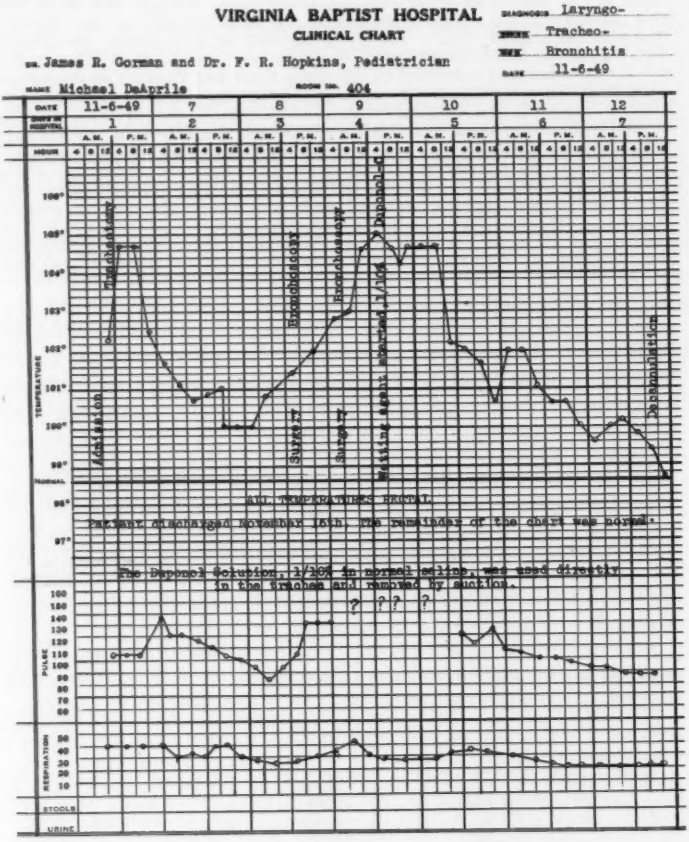
up. Repeating this procedure several times, considerable material was removed, which, when placed on a towel, hardened in about 10 minutes to the consistency of hardened glue. After its removal the tracheotomy tube was reintroduced and the child watched for half an hour and then returned to his room. His temperature had increased from 100 to 102.° I was unable to have slides made but the original hospital chart is shown below.



At 3 A. M. the morning of Wednesday I was called back to the hospital, and the above procedure had to be repeated as a lifesaving measure. This was the morning of the fourth hospital day. The child was receiving large doses of penicillin and highly humidified air in croup tent, and aureomycin.

We did not have a resident at this hospital. My office is five miles and my home seven and one-half miles from the hospital. Fearing the child would die before I could reach him, I called Dr. Fletcher D. Woodward at the University of Virginia and told him my problem. I wanted to send the child there as he has trained residents, but he thought we were doing everything possible. In the discussion with Dr. Woodward, he asked whether I was using saline in the trachea. This, of course, had been done, but it had no effect in this particular case. I asked him if he had ever used a wetting agent in the saline. He said "he had not but thought it would be a good idea." The problem then was to obtain the proper wetting agent. I consulted the U. S. Pharmacopea and found that Duponol-C was listed and approved by the Pure Food and Drugs Commissioner. I am indebted to Mr. N. T. Gorchoff, of the Virginia Specialty Co., for furnishing me with the original supply of Duponol-C which was used in normal saline one-tenth per cent and instilled directly into the trachea through the tracheotomy tube, using a medicine dropper, followed by immediate use of suction, withdrawing fluid mixed with secretions. From the time the solution was started the child began to improve rapidly and was able to cough up the material which we previously had to remove on two occasions. The one-tenth per cent solution of Duponol-C was started at 6 P. M. on Wednesday; on Saturday I was able to decannulate the child, and on Wednesday of the following week was able to discharge the patient.

The cases which present these dry crusts and crusting material in the trachea and bronchi offer a big problem. Cases have died with the bronchoscope in place and in addition to this danger the scope may push the crusts down into the trachea and the main stem bronchi, and the child may die on the table. Dr. Woodward told me he had had one die on the table, being unable to remove the crusts. The small calibre Jackson malleable copper suction tube is a perfectly safe instrument to use through the tracheotomy wound if gently introduced and removed when pressure gauge shows resistance and the secretion or crust removed from the end of tube.

This particular patient had a 10-day hospital stay. The X-rays of chest were essentially negative. The case was treated in consultation with Dr. F. Read Hopkins, pediatrician.

About two weeks later I called Dr. Woodward to report to him the progress made in this case. He said, "I have just left the hospital and have seen a child with laryngotracheobronchitis who needed immediate tracheotomy, and have told the resident to do it, but I will call the hospital and tell them to get some Duponol-C and put it in the water being vaporized." A letter from him a few days later says, "The child admitted

with laryngotracheobronchitis and who apparently needed an immediate tracheotomy, we introduced the wetting agent in water or saline of the vaporizer, at the end of three hours the child was much improved and in five hours was out of danger."

After these two experiences we decided to try its use further with such cases and I wish you would do the same.

Not including the child on whom a tracheotomy had been done, but including Dr. Woodward's case, we now have seven children on whom the drug has been used in the water being vaporized with equally dramatic results and on whom it was not necessary to do tracheotomies. Two cases have been added to the five reported at Memphis.

Becoming interested and knowing a little about wetting agents and the many uses to which they are being put was the reason for my thinking of it in this connection. Proper agents of this type are being used in eye solutions to lower the surface tension and keep the surface moist for a longer period of time.

The fire departments of many cities are using the agents, which in proper dilution are carried in the 250 gallon tanks. This amount of water properly treated will do approximately what 5,000 gallons of water would do.

I wrote to the American Cyanamid Co. regarding wetting agents in common. They promptly sent me a reprint of the work they had done on animals at their Stanford Research Laboratories. They produce the Aerosols O. T. The reprint is rather long, but in the tests they used rabbits, dogs, monkeys and rats. The animals were fed the wetting agent for a period of six months. They state "that although a total of 221 histological preparations of tissue were made from nine rats, six dogs, three monkeys and four rabbits on high dosage were studied, no significant pathology was observed. This is consistent with the observations of Lorenz, Schenkin and Stewart in mice which received the Aerosol O. T. by mouth."

I was also sent their brochure on wetting agents which gives a great deal of information about them and their uses

generally. Aerosol O. T. is Dioctyl Sodium Sulfosuceinate. Duponol-C is Sodium Lauryl Sulphate ($CH_2(CH_2)_{10}CH_2OSO_3Na$) U. S. P. This particular agent or similar ones are being used in shampoo mixtures, tooth pastes, cosmetics, liquid dentifrices and other materials used about the skin.

All of the cases on which the drug was used were not mine, but the agent was used under my direction.

The two and one-half-year-old son of one of our eye, ear, nose and throat men was seen Dec. 24 about 3 P. M. and it looked very much as though a tracheotomy would be needed. The child was in a very small cubical-like room, closed off with heavy drapery. Humidity at 3 P. M. was 65, using an open bucket on a hot plate. At 3:30 the Duponol-C was added, about one-fifth of a teaspoon to the gallon, and in 30 minutes the humidity had increased to 90. By 6:30 P. M. the child was greatly improved and apparently out of danger. Seen Christmas Day, he was running around in his bed, playing with his Christmas toys, and made an uneventful recovery. The room was so moist that the wallpaper had loosened and the doctor told me he would have to redecorate the room.

The important thing is that the moisture is essential and should be maintained. The use of a wetting agent (in these cases Duponol-C) seems to greatly increase the moistening effect, both of the saline one-tenth per cent used directly into the trachea, when the tracheotomy has been done, and in the water being vaporized, whether by steam or actual vaporization. For the comfort of the patient and attendants, the temperature of the room should be controlled, especially where steam is being used. There should be some ventilation of the room.

Several of our drug stores now stock Duponol-C. Whether this is the best agent to be used remains to be seen and I hope that you gentlemen who have at your disposal laboratory and clinical facilities will use this suggestion and make further reports on studies of its usefulness. Anything which is helpful in laryngotracheobronchitis will be worthwhile.

In talking with Dr. Woodward over the telephone, Feb. 17, he said they had had several cases in which the drug had been used by vaporization and they had been able to avoid tracheotomy on all of them and that there would be subsequent reports on the use of the drug.

1108 Allied Arts Building.

In Memoriam

FRANCIS R. PACKARD, M.D.,
1870-1950.

Dr. Francis R. Packard, prominent medical librarian and pioneer otolaryngologist, died April 18 at Pennsylvania Hospital at the age of 80.

Born in Philadelphia, the son of Dr. John Hooker and Elizabeth Wood Packard, he graduated from the University of Pennsylvania in 1889 and three years later received his medical degree there.

Dr. Packard was first assistant surgeon of the Second Pennsylvania Volunteers during the Spanish-American War, and in World War I was center consultant for the district of Paris.

In 1930, Dr. Packard was elected president of the American Laryngological Association; in 1931, he was president of the College of Physicians of Philadelphia, and in 1936 was president of the American Otological Society. He was also a former president of the Musical Fund Society of Philadelphia and associated with various library groups. He was a member of the Academy of Stomatology of Philadelphia, a member of the Philosophical Society, University Barge Club, Franklin Inn, Philadelphia Shakespeare Society, Wistar Association and the Historical Society of Pennsylvania.

He was the proud owner of one of the largest medical libraries in this country and the author of many formidable papers in his specialty, but his outstanding contribution to otolaryngologic literature was perhaps his History of Medicine in the United States. During the early years of THE LARYNGOSCOPE, Dr. Packard was quite active on its editorial staff and was a frequent contributor to its pages.

As honorary keeper of the archives of Pennsylvania Hospital, he had at his command valuable data from which he compiled a history of the hospital from 1751 until 1918.

He is survived by four daughters: Mrs. E. Perot Bissel, Jr., of Haverford; Miss Elizabeth Packard, of Philadelphia; Mrs. Peyton R. Biddle, of Rosemont, and Mrs. John H. Rhein, of Seaford, Del.

BOOK REVIEWS.

Fundamentals of Otolaryngology. A Textbook of Ear, Nose and Throat Diseases. By Lawrence R. Boies, M.D., Clinical Professor of Otolaryngology, Director, Division of Otolaryngology, University of Minnesota, and associates Charles E. Connor, M.D., Anderson C. Hilding, M.D., Jerome A. Hilger, M.D., John J. Hochfilzer, M.D., Conrad J. Holmberg, M.D., Kenneth A. Phleps, M.D., Robert E. Priest, M.D., and George M. Tangen, M.D. 433 pages with index and 184 illustrations. Philadelphia and London: W. B. Saunders Co., 1949.

This is an excellent book for undergraduate students and as a reading text in postgraduate courses. Dr. Boies is well qualified to write such a book. He states the objectives of the book in his preface and these objectives are well fulfilled.

This volume should be in the library of all medical schools.

T. E. W.

The Comparative Anatomy and Physiology of the Larynx. By V. E. Negus, M.S., F.R.C.S., Surgeon to the Ear, Nose and Throat Department of King's College Hospital, London. With foreword by Sir Arthur Keith. First edition. 230 pages with index and 191 figures. New York: Grune & Stratton, Inc., 1949.

This book, as the author states in his preface, is a condensation of his previous work, "The Mechanism of the Larynx." It is an excellent and complete work, profusely and beautifully illustrated. The author leaves no phase of the anatomy or physiology of the larynx untouched.

Particularly valuable for the clinician is the chapter on the anatomy of the human larynx and the appendices. This book should be in the library of every department of laryngology. T. E. W.

The Nose. An Experimental Study of Reactions Within the Nose in Human Subjects During Varying Life Experiences. By Thomas H. Holmes, M.D.; Lester H. Hofheimer, Research Fellow in Medicine; Helen Goodell, B.S., Research Fellow in Medicine; Stewart Wolf, M.D., Associate Professor of Medicine; Harold G. Wolf, M.D., Professor of Medicine (Neurology), Cornell University Medical College, New York, N. Y. With a foreword by Warfield T. Longcope, M.D., Professor Emeritus of Medicine, The Johns Hopkins Medical School, Baltimore, Md. 154 pages with index and 37 illustrations. Springfield, Ill.: Charles C. Thomas, Publisher, 1950. Price $4.50.

This is a most interesting treatise on nasal pathologic changes resulting from situational threats. The subject is too frequently overlooked by rhinologists. Of particular interest and importance are the histologic and cytologic findings reported by the authors. The concept that eosinophiles in secretions and tissues is pathognomonic of allergy must, in the light of their observations, be seriously questioned.

The book makes fascinating reading and is strongly recommended to all otolaryngologists. T. E. W.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer; Maico UE-Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic (Crystal Receiver) Model 101; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Philip E. Meltzer, 20 Charlesgate, West Boston 15, Mass.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-22, 1950.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Ralph A. Fenton, 906 Medical Arts Bldg., Portland, Ore.
Secretary: Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 23-24, 1950.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Robert C. Martin, 384 Post St., San Francisco 8, Calif.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester, N. Y.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 25-27, 1950.

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Secretary: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 17-20, 1950.
Meeeting: Palmer House, Chicago, Ill., October 3-6, 1950.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles, Calif.
President-Elect: Dr. Derrick Vail, Chicago, Ill.
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Meeting: Palmer House, Chicago, Ill., October 8-14, 1950.

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Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia, Pa.

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Meeting: Mark Hopkins Hotel, San Francisco, Calif., May, 1950.

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Secretary-Treasurer: Dr. Victor Goodhill.
Chairman of Section on Ophthalmology: Dr. Dennis V. Smith.
Secretary of Section on Ophthalmology: Dr. Carroll McCoy.
Chairman of Section on Otolaryngology: Dr. Howard P. House.
Secretary of Section on Otolaryngology: Dr. Edwin Scobee.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

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President: Dr. G. M. Billings, Morganton, N. C.
Secretary and Treasurer: Dr. MacLean B. Leath, High Point, N. C.
Time and Place: Sept. 11-13, Hendersonville, N. C. Jointly with the South Carolina Society of Ophthalmology and Otolaryngology.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. David R. Higbee, 3245 4th St., San Diego, Calif.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco, Calif.
Meeting: St. Francis Hotel, San Francisco, Calif., May 28-31, 1950.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

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Chairman-Elect: Dr. Francis LeJeune, Ochsner Clinic, New Orleans, La.
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